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# Psychometric Study of the Mahan and DiTomasso Anger Scale (MAD-AS) in an Outpatient Cardiac Sample

Kimberly S. D'Andrea
*Philadelphia College of Osteopathic Medicine*, drdandrea@yahoo.com

Philadelphia College of Osteopathic Medicine

Department of Psychology

# A PSYCHOMETRIC STUDY OF THE MAHAN AND DiTOMASSO ANGER SCALE (MAD-AS) IN AN OUTPATIENT CARDIAC SAMPLE

By Kimberly S. D'Andrea, Psy.D.

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**Dissertation Approval**

This is to certify that the thesis presented to us by Kimberly S. D'Andrea on the 11th day of October, 2004, in partial fulfillment of the requirements for the degree of Doctor of Psychology, has been examined and is acceptable in both scholarship and literary quality.

**Committee Members' Signatures:**

**Robert A. DiTomasso, Ph.D., ABPP, Chairperson**

**Harry J. Morris, D.O., M.P.H.**

**Steven Godin, Ph.D., M.P.H.**

**Robert A. DiTomasso, Ph.D., ABPP, Chair, Department of Psychology**

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Abstract

Psychometric evaluation of the Mahan and DiTomasso Anger Survey (MAD-AS) analysis in an outpatient cardiac population is herein described. The MAD-AS is a newer instrument that measures the cognitive, affective, and behavioral components of anger and is beneficial in that it is brief. One hundred fifty cardiac outpatients and one hundred fifty non-cardiac dental patients were administered a demographic form that requested information about cardiac risk factors, the MAD-AS, and the STAXI-2, a widely used valid and reliable measure of state and trait constructs of anger. The MAD-AS factor structure, construct validity, internal consistency, and reliability were examined and found to support the instrument. Group differences were hypothesized to exist in regards to anger; these differences were not statistically significant. However, when age and several risk factors were controlled for, group differences were detected in the expected trajectory in MAD-AS scores but not in STAXI-2 scores. The results described in this study support the reliability and validity of the MAD-AS.

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# Chapter 1

## Introduction

According to the American Heart Association (2004), coronary heart disease (CHD) has been the greatest killer of Americans each year since the early 1900's. Approximately every 26 seconds one person will experience a cardiac event, and each minute one person will die from it. In fact, about 42% of individuals who experience a cardiac event will die from it (AHA, 2004). CHD was responsible for 1 in 2.5 deaths in 2001; however, identifying traditional genetic and lifestyle risk factors for heart disease does not explain approximately 50% of cardiac diagnosis each year (Futterman & Lemberg, 1998; Hennekens, 1998). Thus, the search for viable correlates of heart disease has become vastly important.

One of the most promising psychological correlate of heart disease is anger, hostility, and aggression (e.g., Fredrickson, Maynard, Helms, Haney, Siegler, & Barefoot, 2000; Houston, 1994; Suarez, Kuhn, Schanberg, Williams, & Zimmering, 1998; Suls & Wan, 1993). These factors have emerged from early research on a characteristic behavior pattern termed "Type A Behavior Pattern" (also referred to as "Type A" or TABP).

As far back as the1950's, researchers were interested in investigating the psychological links to CHD and identified the TABP, which refers to an individual who is very competitive, often impatient, has a strong sense of time-urgency, and who experiences easily aroused hostility and anger (Friedman & Rosenman, 1959; Rosenman et al., 1964). Since then, much effort has been put forth to elucidate further the

mechanisms involved in the etiology, progression and maintenance of heart disease. When some studies, using more stringent research methods, did not find support for the TABP/CHD correlation, research became focused in the 80's and 90's on anger, hostility, and aggression as the potent components of TABP most associated with CHD (as cited in Ravaja, Kauppinen, & Keltikangas-Jarvinen, 2000).

Although many of the pathways that may lead to CHD have been illuminated, agreement on how to best measure anger, hostility, and aggression has not been established. Coupled with that dilemma is the fact that most measurement instruments are quite lengthy. Thus, the development of a brief, well-validated instrument that successfully taps the cognitive, affective, and behavioral aspects of anger constructs is needed. Such an instrument may potentially be implemented as a screening tool for use in the primary care setting to identify at-risk individuals. Given the devastating financial and human impact CHD has on our society, the health of a great number of people may be positively affected if individually tailored treatment plans are implemented as a result of this measure.

## Purpose

The purpose of this investigation is twofold. The principal purpose of this study is to add to the normative data of the Mahan and DiTomasso Anger Survey (MAD-AS), a newer instrument that measures the cognitive, affective, and behavioral components of anger. One of the chief benefits of this instrument is that it is relatively shorter than other surveys. Therefore, the primary goal of this study is to evaluate further psychometric

properties of the MAD-AS as a succinct instrument to measure the constructs of anger in a cardiac and non-cardiac outpatient population. Strong psychometric properties may introduce the possible clinical use of the MAD-AS as a screening tool when early interventions may positively impact the health of many at-risk Americans.

The second purpose of this investigation is to contribute to the body of research elucidating significant psychological correlates of coronary heart disease.

## Rationale

### *Heart Function*

The human heart, whose function is to pump blood throughout the body, is a very strong muscle about the size of an adult fist. Blood passes from the right side of the heart into the lungs where it becomes oxygenated and then returns to the left side of the heart. From the left side of the heart, blood is dispersed throughout the circulatory system.

The circulatory system is made up of the heart, lungs, arteries, arterioles, capillaries, and venules. These structures are an incredibly long and intertwined group of tubes that deliver oxygen rich blood to all areas of the body. They also clean out cell waste products by filtering them through the lungs, liver, and kidneys. The blood travels throughout the circulatory system when the heart muscle contracts its four chambers in a specific sequence. The contractions are governed by an electrical impulse that originates in the right atrium, which is also called the sinus node. The electrical impulse discharges a signal that causes the heart to beat (Brunwald, 1992). However, all too often, a

multitude of factors may prevent the heart from performing its job properly, resulting in varying forms of coronary heart disease (CHD).

*Coronary Heart Disease*

The broad term of cardiovascular disease (CVD) is composed of several forms of heart disease including hypertension, congestive heart failure, stroke, congenital heart defects, and coronary heart disease (CHD). CVD has been the has been the number one killer of Americans since the 1900's, claiming more lives each year than the other five major causes of death combined, including cancer, respiratory diseases, accidents, diabetes, flu, and pneumonia (American Heart Association, 2004). For those under the age of 75, more than half of all cardiovascular events are due to CHD, which makes this the single largest killer of Americans (AHA, 2004).

CHD includes heart attack also called myocardial infarction (MI), angina pectoris (chest pain) or both. Every 26 seconds someone will suffer a coronary event and every minute someone will die as a result; about 42% of MI occurrences result in death. Approximately 80% of deaths due to CHD in those under age 65 occur during the first attack. 25% of men and 38% of women will die within one year of having an MI and half of those under 65 who have had an MI will die within 8 years of the attack. What is even more troubling is the fact that 50% of men and 64% of women who have suddenly died of CHD did not have any symptoms (AHA, 2004). This fact highlights the importance of knowing one's risk factors.

According to the AHA (2004), about two-thirds of patients do not make a complete recovery after a heart attack, thus, CHD is also the major cause of premature, permanent disability in the United States workforce, accounting for 19% of Social Security Administration disability allowances; 10.7 billion dollars was paid to Medicare beneficiaries for CHD in 1999. The direct and indirect cost of CHD alone is 133.2 billion dollars and for all cardiovascular diseases, the cost has been estimated at a staggering 368.4 billion dollars. Direct costs include cost of physicians and other health care professionals, hospital and nursing home services, home health care, medication, and medical equipment. Indirect costs consist of lost productivity that results from morbidity and mortality. For comparison sake, the 2003 estimated cost both direct and indirect for all cancers is 189 billion.

The lifetime risk of developing CHD when an individual reaches 40 years of age is 49% for males and 32% for females (AHA, 2004). For comparison sake, it was also calculated that at birth, a person's long term probability of dying from CHD is 47%; however, the probability of dying from cancer is 22%, 3% for accidents, and 0.7% for HIV (AHA, 2004). Given the tremendous impact CHD has on American society in financial terms and more importantly in terms of human suffering, it is apparent that identifying all potent risk factors of CHD is essential to the health and well being of a tremendous number of individuals.

*Biological and Lifestyle Risk Factors of CHD*

Several biological and lifestyle risk factors of CHD have been established by the American Heart Association; these include premature family history, male sex (female risk is almost equal to male after menopause), smoking, overweight and obesity, physical inactivity, hypertension, high cholesterol, and diabetes. Unless otherwise noted, all biological and lifestyle risk factor statistics were derived from the Heart Disease and Stroke Statistics – 2004 Update (AHA, 2004).

*Premature family history.* The CHD risk factor that cannot be modified is premature family history. That is, a first-degree relative such as a sibling or parent, younger than 55 for men and younger than 65 for women, who have had CHD is considered a genetic independent risk factor for the disease and is not modifiable.

*Male sex.* American men under the age of 74 have a greater risk of CHD than do women in that same age category. The incidence of CHD in women lags behind men by 10 years. The lag time is attributed to hormone changes resulting from menopause. CHD rates in women after menopause are over 2 times higher than in women before menopause. Therefore, male sex status independently places middle-age men at risk for CHD.

*Smoking.* One of the lifestyle risk factors highly related to CHD is smoking. Although tobacco use has declined more than 40% since 1965, smoking continues to kill

far too many Americans. An average of 442,398 people have died from 1995 through 1999 from smoking-related illnesses and over 33% of those deaths were cardiovascular related. Even if an individual does not smoke, the health effects from tobacco can cause death. Approximately 35,000 nonsmokers die annually from CHD because they were exposed to second-hand smoke at home or at work. Including smoking-attributable lost productivity costs, neonatal medical expenses, and medical expenditures, smoking costs more than 157 billion dollars per year.

*Overweight and obesity.* One risk factor of CHD that has become a topic of concern among many healthcare professionals is overweight and obesity. Americans are becoming larger at an alarming rate. The number of total calories from fat in the American diet has decreased over the past decade, however, the total daily caloric intake has increased, contributing to the swift increase in obesity. Approximately 300,000 adults die every year from obesity-related causes. Overweight and obesity obese is determined by calculating one's Body Mass Index (BMI), taking weight in kilograms and dividing that number by height in meters squared ($kg/m^2$). Overweight in adults is defined as a BMI of 25 – 29.9; obesity is defined as a BMI of 30 or greater; and extreme obesity is defined as a BMI of 40 or greater. These numbers have risen dramatically in recent years. For instance, the prevalence of obesity since 1991 has increased a startling 75%. In a similar time period from 1988 through 1994 the prevalence of overweight individuals has increased from 55.9% to 64.5%; obesity prevalence has gone from 22.9% to 30.5%, and extreme obesity in the same time period has risen from 2.9 to 4.7%. Being

overweight and being obese are also costly conditions, estimated to cost about 100 billion dollars annually. The impact on one's lifespan is also greatly influenced. For instance, the lifespan of a 20 year old extremely obese white man whose BMI is greater than 45 is reduced by an estimated 13 years. Thus, being overweight and being obese are clearly significant, modifiable threats to one's health status.

*Physical inactivity.* A major modifiable risk factor that plays a role in CHD mortality is physical inactivity. Lack of physical activity is associated with a relative risk of CHD comparable to smoking, hypertension, and high cholesterol. Data from 2000 – 2001 indicated that of Americans over the age of 18, 54.6% were not active enough to meet physical activity recommendations. Moderate activity is defined as engaging in activities such as walking or bicycling at least 30 minutes on 5 days of a 7 -day period. Vigorous activity is defined as activities that produce heavy breathing and sweating for at least 20 minutes on 3 days or more of a 7 -day period. In the year 1997-1998, 38.3% of Americans over age 18 reported that they did not get any physical activity at all; some form of physical activity was reported by 61.7%, and light to moderate physical activity was reported by 22.7%. The annual cost related to physical inactivity is 76 billion dollars per year.

*Hypertension.* Hypertension (High blood pressure or HBP) is defined as a systolic pressure of 140 mm Hg and over, or a diastolic pressure of 90 mm Hg and over or both. There is also a condition termed "prehypertension" that refers to a systolic

pressure between 120-139 mm Hg, a diastolic pressure between 80-89, or both. One of four American adults has hypertension and approximately 22% have prehypertension. 30% of those with hypertension do not know they have it, 34% are on medication for it and have it controlled, 25% are on medication but do not have it under control, and 11% are not on medication. Before the age of 55 a higher percentage of men have hypertension, from 55-74 women have a somewhat higher percentage, and over the age of 75 women have a much higher percentage of hypertension than men.

Black Americans are disproportionately afflicted with this condition. The prevalence of hypertension in black Americans is the highest in the world. As compared with white Americans, black Americans have a 1.5 times greater rate of death from heart disease.

About half of those who experience their first heart attacks have blood pressures that are 160/95 mm Hg or higher. Hypertension was the primary or contributing cause of death in over 250,000 deaths in 2000. The death rate from hypertension rose 36.4% from 1991-2001 and the number of deaths rose 53%. The direct and indirect costs for 2004 are estimated to be 55.5 billion dollars.

*High cholesterol.* Total blood cholesterol is determined by the measurement of serum cholesterol in one's blood. Levels of 200-239 mg/dL are considered borderline high risk in adults and levels above 240 mg/dL are considered high risk. However, total cholesterol levels give limited information about CHD risk; a consideration of the breakdown of HDL and LDL gives more valuable information. HDL is considered the "good" cholesterol and the higher the number the better. 40 mg/dL and below is

considered too low and is a CHD risk factor. Conversely, LDL is considered the "bad" cholesterol and the lower the better. A level of 130-159 mg/dL is considered borderline high, 160-189 mg/dL is considered high, and over 190 mg/dL is considered very high. Adults whose good cholesterol is low and whose total cholesterol is high have the greatest risk of a heart attack. HDL is a very potent factor in assessing health status because men with lower than 37 mg/dL are at high risk of a heart attack regardless of their total cholesterol level. In fact, a person who has high total cholesterol still carries the low risk of a heart attack if even if he or she has high HDL.

Management of lipids is problematic and adherence to treatment regimen is a major factor. If the population had a mere 10% decrease in total cholesterol, there would be an estimated 30% reduction in CHD. But only 50% of those who meet criteria for lipid treatment for CHD risk reduction receive it. Fewer than 50% of the highest risk adults who have symptomatic CHD are receiving treatment. Of those who are prescribed lipid modifying treatment only about 50% continue to take it six months after it is prescribed; this is a dilemma because it takes up to a year before the medications change lipid profiles.

*Diabetes.* Diabetes is defined as a fasting blood glucose level greater than 126 mg/dL. The rate of diagnosed diabetes continues to rise in America. In fact, the prevalence of diabetes has increased 61% since 1990. During the year 2000, of the 4.4 million adults who had diabetes, 2.9 million were diagnosed with CHD. CHD death rates for diabetic adults are 2 to 4 times greater than non-diabetic adults, and up to three fourths of adults diagnosed with diabetes die from a heart or blood vessel disease. .

However, accounting for all of these aforementioned risk factors still leaves a great percentage of CHD unexplained (Kannel & Schatzkin, 1983). Therefore, considerable effort has been made in identifying other salient psychological risk factors. Research in this area began with the identification of TABP and has progressed through the years to identify more specifically the most fruitful of these risk factors: anger, hostility, and aggression (Smith, 1992).

*Psychological Factors and CHD*

The progression to heart disease does not occur quickly and it is not an inevitable result of the aging process. It is often, but not always, without symptom at the onset of atherosclerosis; it is often followed by warning signs such as angina, and then progresses to events such as heart attack or sudden death. The average age of a first heart attack is 65.8 for men and 70.4 for women (AHA, 2004). The disease process generally spans many years of risk factors interacting with various biochemical, immune inflammatory and hemodynamic processes before symptoms manifest themselves (Krantz & McCeney, 2002). It is a progressive situation because the mind and body continually interact

through the system's autonomic nervous system, musculoskeletal system, and the psychoneuroendocrine system (Buselli & Stuart, 1999); therefore, risk factors and disease operate in a bi-directional manner. Thus, considering one's psychological risk profile utilizing the biopsychosocial model (biological factors, psychological factors, and social/environmental factors) are as important in a complete picture of health status as are an assessment of only genetic and lifestyle risk factors.

*Type A Behavior Pattern (TABP)*

Early research studies that examined psychological links to CHD (Friedman & Rosenman, 1959; Rosenman et al., 1964) identified a specific characteristic pattern that appeared to be related to CHD; this was termed Type A Behavior Pattern or TABP. This phrase became a highly publicized buzz- word that is still today a well- known term. It refers to an individual who is extremely competitive, often impatient, has a strong sense of time-urgency, and is often hostile and angry. TABP individuals display chronic activation, hence they are keyed up and stay keyed up most of the time. They are also multiphasic, meaning they usually do more than one thing at a time (Wright, 1988). This concept received considerable support and the correlational association was a strongly held notion for many years. For instance, a quantitative meta-analysis determined TABP to be significantly associated with CHD (Booth-Kewley & Friedman, 1987), and some studies have been able to demonstrate that TABP individuals have more than 2 times the incidence of CHD (Friedman, et al., 1986; Haynes, Feinleib, & Kannel, 1980). However,

over time, because several studies using more stringent research methods (Hearn, Murray, & Luepker, 1989; Leon, Finn, Murray, & Bailey, 1988), did not find support for TABP, some researchers advocated the abandonment of the TABP concept altogether (e.g. Conduit, 1992). Instead, more focused investigations began to isolate the most toxic aspects of TABP that were responsible for the link to CHD. The factors that began to emerge as being significant are anger, hostility, and aggression.

*Anger*

The emotion of anger has been a rich topic of research for decades. Theories abound about the complexities of the anger experience. Early theorists debated one of the most fundamental aspects of anger: how does one come to experience the emotional feeling and physical reaction of anger? This debate began in earnest when psychologist William James collaborated with physiologist Carl Lange and concluded that aversive stimuli produces distinct physical reactions (e.g. increased heart rate and respiration, tightened facial muscles) particular to each emotion and that once the individual perceives these physical sensations they then attribute them to the feeling of anger. Simply put, the James-Lange theory (as it has been termed) states that physical sensation precedes the emotion (Lange & James, 1922).

Conversely, two physiologists, Walter Cannon and Philip Bard came to the opposite conclusion. Their theory (Cannon, 1929) posited the idea that physiological reactions were not specialized to each specific emotion. One could experience sweating

and a racing heart for various reasons such as love, anger, or fear. They believed that bodily sensations are a result of the body's "fight or flight" response (the neuroendocrine activation that prepares the body to flee or deal with severe bodily injury) and that these sensations are more universal. In the Cannon and Bard conceptualization, feelings and sensations occur simultaneously. Accordingly, research has continued throughout the years in attempts to understand the mechanisms involved in the experience of anger.

Anger is often perceived as a negative emotion (Watson & Clark, 1991) that is undesirable to express. Indeed, uncontrolled control anger can lead to crime, violence, and other destructive behavior. The reality is that anger is a very important adaptive function, a normal response to a threatening situation. When such a situation occurs, anger can be positively directed to guide our actions. If not for the fight or flight response, the human race would not have survived. If an individual could not fend off or flee from an attacking lion that person would be eaten, thus, the fight or flight response has served us well.

In a social situation anger is conceptualized as just as adaptive in responding to a threat (Beck, 1999). One could perceive a verbal assault as being just as deleterious as a physical assault and prepare to protect oneself. These are all positive and appropriate signals that guide one's actions and are part of everyday life. However, distinguishing between healthy and toxic anger behaviors has revealed interesting results. For instance, a systematic literature review of prospective studies from 1980 to 2000 has found empirical evidence stating that emotions such as anger are linked to CHD (Tennant & McLean, 2001). On the other hand, the principal aspect of the anger experience that is

most health threatening continues to be debated. For example, it has been proposed that it may be the frequency and persistency of physiologic activation that is more related to disease progression (Herrald & Tomaka, 2002) and not the mere experience or severity of anger. It has also been suggested that the outward, chronic, full-blown expression of anger is pathogenic, not simply the experience or suppression of the anger (Siegman & Snow, 1997). In support of the anger expression theory, it has been concluded that patients with CHD who expressed anger outwardly and who experienced low social support were at a highly increased risk of disease progression, a risk that was independent of other risk factors and medications (Angerer, Siebert, Kothny, Muhlbauer, Mudra, & von Schacky, 2000). Hence, the search for clarity in the role through which emotions relate to CHD has continued and has pointed to anger, hostility, and aggression constructs as salient factors.

When Helmers et al. (1993) examined hostility and CHD risk, they distinguished between various types of hostility (high, low, defensive) to look at possible differences. It was observed that those who were categorized as high hostile showed the most severe ischemia, therefore, the most negative health prognosis. When women were studied to explore the relationship of hostility to lipid profile, those with high antagonistic hostility scores (as opposed to cynical and neurotic hostility) had higher cholesterol levels (Suarez, Bates, & Harralson, 1998). Moreover, a meta-analysis examining hostility and physical health found that hostility has been observed to be an independent risk factor for CHD (Miller, Smith, Turner, Guijarro, & Hallet, 1996). This is suggestive of a pathophysiolgical mechanism between hostility and CHD which is similar to anger.

There is no doubt that the conceptual clarity provided by the STAXI concerning anger and hostility have been invaluable in elucidating the link to CHD, specifically in delineating the state vs. trait dimensions of these concepts.

*State/Trait Distinction*

Research on anger and hostility has been confusing because of vague definitions of the constructs and the interchangeable use of terms. Spielberger (1988; Spielberger, Reheiser, & Sydeman, 1995) made an important distinction when he and his colleagues separated the concept of state anger from trait anger and developed the State-Trait Anger Expression Inventory (STAXI) (Spielberger, 1988) and its updated version, the STAXI-2 (Spielberger, Reheiser, & Sydeman, 1995). Spielberger holds that the state/trait distinction is essential in understanding what constitutes an emotional state that is situationally driven (state) versus an enduring predisposition (trait) to respond in a certain manner. State anger is conceptualized as a transitory emotion with varying intensities. Trait anger is conceptualized as an enduring pattern of responding with higher intensity of anger to problematic situations, accompanied by the activation of the neuroendocrine system and autonomic nervous system. This distinction has been crucial in understanding the concepts of anger, aggression and hostility and the possible relationship to CHD.

Deffenbacher and colleagues (Deffenbacher et al., 1996) posit that if one is to understand trait anger as an anger-prone disposition, then five theoretical predictions follow for an individual with high trait anger. These predictions include: (1) Elicitation

Hypothesis: There is a tendency to be angered easily. (2) Intensity Hypothesis: There is a tendency to respond more intensely when provoked. (3) Negative Expression Hypothesis: There is a tendency to express anger in less adaptive ways, which leads to more frequent anger suppression and negative anger suppression of anger, as well as less adaptive coping. (4) Consequence Hypothesis: Due to more frequent and intense anger and fewer coping skills, there is a tendency to encounter more frequent anger-related consequences. (5) Discrimination Hypothesis: There is a tendency to relate more easily to anger-related constructs than any other emotion. Eight studies were conducted (Deffenbacher et al., 1996) to test these hypotheses and all five theories were supported. Evidently, high trait angry individuals deal with negative fallout due to negative expressions of anger, increased volatility, and unconstructive methods to deal with situations. One of the most dangerous consequences of this disposition is the development of CHD. Several large-scale research studies have confirmed this hypothesis (Chang, Ford, Meoni, Wang, & Klag, 2002; Williams, Paton, Seiger, Eigenbrodt, Nieto, & Tyroler, 2000).

Another benefit of the STAXI was that it made a clear distinction between anger aggression and hostility. It is suggested that deficiencies in measuring such concepts are due to overlapping concept definitions (Arthur, Garfinkel, & Irvine, 1999); this was partially responsible for the resulting abandonment of the TABP concept. The STAXI delineated the fact that anger constitutes an emotional state that varies from mild aggravation to intense rage with accompanying physiologic responses; hostility is composed of negative attitudes including meanness and viciousness, and aggression includes aggressive and vindictive behaviors. In addition, anger is generally

conceptualized as a feeling and hostility and aggression involves negative attitudes and behavior. Thus, it is understandable that research into anger is often confounded because the constructs of anger, hostility, and aggression are intrinsically related yet are very unique. Barefoot (1992) defined hostility as the predisposition towards antagonistic behavior, with a preponderance of angry and cynical thoughts and feelings. Because hostility was exposed as a possible risk factor of CHD and separate from TABP, it has prevailed as a critical link to the disease (Dembroski, MacDougall, Costa, & Grandits, 1989; Williams, Haney, Lee, Kong, Bluementhal, & Whalen, 1980). High hostility scores were found, in early research, to be a factor in significant increases in clinical coronary events (Barefoot, Dahlstrom, & Williams, 1983; Shekelle, Gale, Ostfeld, & Paul, 1983) separate from TABP. Since that time, more recent investigations have been conducted and these investigations have found hostility, along with anger and aggression, to be a potent health threat.

*Anger, Hostility, and Aggression (AHA! Syndrome)*

The anger - hostility – aggression components within TABP began to emerge as the most salient constructs that were predictive psychological correlates of CHD; these are referred to as the AHA! Syndrome. One study found that the AHA! Syndrome was directly related to total serum cholesterol and low-density lipoprotein (LDL) in men (Richards, Hof, & Alvaregna, 2000). LDL is also known as "bad cholesterol" and as previously mentioned, it is healthy to have this at a low level; however, high-density

lipoproteins (HDL) are commonly referred to as "good cholesterol" and this number should be high for heart health. These findings suggest that the disposition (trait) to express an angry affect may be a risk factor for developing CHD via an unfavorable lipid profile. Although the relationship between serum lipids and CHD remains quite complex, Van Doornen (1997) concluded that there is a relationship between the two factors, even when mediating lifestyle variable are controlled. Without the STAXI state/trait anger conceptualization, the AHA! Syndrome in relation to CHD may not have been identified. This distinction has directed the vast amount of research into psychological correlates of CHD.

*Related Research*

*The Precursors study.* The Johns Hopkins Precursors Study, a large prospective longitudinal study of 1,055 males followed participants for a period of time from 32 to 48 years (Chang, Ford, Meoni, Wang, & Klag, 2002). The original study began in 1946 and consisted of 1,337 medical students who graduated from medical school in the years from 1948 through 1964. Student participants underwent physical examinations, completed personal and family histories, and completed questionnaires on health status, behaviors, and stress reactions. Every 5 years the participants were followed up via mailed surveys, with a 90% response rate. The vital status of the participants is known for over 99% of the entire cohort. When women, those who did not respond and those who reported heart disease before graduation were excluded, 1,055 male participants remained for analysis.

Anger was assessed using the Habits of Nervous Tension Questionnaire (Thomas, 1977). This measure asks "Whenever you find yourself in situations of undue pressure or stress, how do you usually react?" and then provides 27 items to check. Three factors were identified through factor analysis as anger: 1) expressed or concealed anger, 2) irritability, and 3) gripe sessions. The validity of this measure was corroborated by its significant correlation with the Multidimensional Anger Inventory (Siegel, 1986) total score and subscale scores of Anger-In, Anger-Arousal, Hostile Outlook, and Range of Anger-Eliciting Situations. When the Habits of Nervous Tension Questionnaire was administered again in 1992, approximately 43 years after graduation, the 3 previously described items remained clustered, suggesting that the same anger constructs were assessed in both administrations. Outcome analysis was performed using premature CHD (before age 55) and total CHD (defined as MI, sudden death, angina, ischemic heart disease, and any other heart disease that entailed heart bypass surgery, coronary interventions, hypertensive heart disease, heart failure, cerebrovascular disease, atherosclerosis, aortic aneurysm, peripheral vascular disease, and arterial embolism). High and low anger as the independent variable, age, and the relationship to premature and total CHD was analyzed. A substantial effect was observed between high anger response to stress in early life and premature CHD later in life; this was true especially MI in men, supporting the anger correlate of CHD.

*ARIC study.* The Atherosclerosis Risk in Communities (ARIC) study conducted by the National Heart, Lung, and Blood Institute (NHLB) is a large ongoing population-

based prospective study of CHD, utilizing both cohort and surveillance components in the design (ARIC Investigators, 1989). Participants were 14,348 white and black males and females between the ages of 45 and 64 at baseline data collection from 1987 to 1989. The U.S. communities chosen to participate using random probability sampling procedures were Jackson, Mississippi; Washington County, Maryland; Minneapolis, Minnesota, and Forsyth County, North Carolina. Annual morbidity and mortality surveillance, as well as triennial clinical examinations have taken place.

A study was conducted on the follow-up period from1990 to 1992 to the end of 1995; the study examined the incidence of CHD events of the ARIC participants (Williams, Paton, Seiger, Eigenbrodt, Nieto, & Tyroler, 2000). Of the 14,348 participants, 13,208 were free of CHD at the second follow-up period. Once exclusions for missing and/or incomplete data were made, 12,986 participants remained to be analyzed. A CHD incident was operationally defined as having an acute MI/ fatal CHD, silent MI, or cardiac revascularization procedures. All CHD events were validated by documented clinical evidence (e.g. ECG's and cardiac enzymes) and by coroner or medical examiner's reports. Trait anger was assessed using the Spielberger Trait Anger Scale (Spielberger, Jacobs, Russell, & Crane, 1983). The overall trait anger score (from minimum of 10 to a maximum of 40) was stratified into three levels; 10 – 14 low trait anger, 15 – 21 moderate trait anger, and 22 – 40 high trait anger. Of all the traditional risk factors for CHD (e.g. smoking, diabetes, overweight, hypertension), only hypertensive status was found to correlate statistically with high trait anger scores. It was found that high trait anger placed normotensive participants at significant risk for CHD

death and morbidity, independent of established risk factors. Using proportional hazard regression analysis to calculate Hazard Ratios (HRs), the study found that high trait anger participants were 2.61 more likely to experience a CHD event than low trait anger participants. Participants who were in the moderate range for trait anger had a 40% greater risk for a CHD event compared with low trait anger participants. Also, it was found that the risk of experiencing a "hard" event (acute MI/fatal CHD) was almost 3 times greater in high trait anger participants compared with low trait anger counterparts. The risk of high trait anger leading to CHD in hypertensive participants did not prove to be statistically significant. Thus, the researchers analyzed whether or not blood pressure medication use (beta-blockers, calcium channel blockers, ACE inhibitors, diuretics) or other medications (aspirin, other antihypertensives, antianxiety medications, and antidepressants) could have impacted the results. It was found that hypertensive status, regardless of participants' use or non-use of the aforementioned medications, did not correlate with the anger – CHD risk. This confounds the anger/CHD correlation because it was found to be significant only for those with normal blood pressure. Hence, the biological pathways involved in the anger – CHD process continue to be quite complex and thus far elusive.

*Determinents of Myocardial Infarction Onset study.* The Determinents of Myocardial Infarction Onset Study (Onset Study) assessed whether or not anger outbursts can trigger a nonfatal acute MI (Mittleman, et. al, 1995). The study utilized a case-crossover design, a new epidemiological technique developed for the study. In this

design, each case serves as is its own control. The design allows researchers to assess change in health risk because of exposure to a brief but hazardous period based on the patients' past exposure experiences. The Onset Study, which was conducted at 45 sites in the Boston area between August 1989 and March 1993 included 1,623 MI patients. Interviewers were trained in person, with a manual, with a video, and with feedback from the study coordinator. In addition, one-third of the interviews were audio taped and checked for accuracy. The interviewers were not told about the hypothesis concerning the "hazard period" in order to ensure non-biased interviewing. Information was gathered about the time, place, pain and other symptoms, estimation of frequency of exposure to anger in the preceding year, and the timing and intensity of anger, as well as other potentially hazardous factors in the preceding 26 hours before the onset of the MI. Anger was assessed using an anger onset scale and the state anger subscale of the State-Trait Personality Inventory. The ratio of exposed frequency and expected frequency (from the control information) of the hazard period was calculated to determine an odds ratio of relative risk. The anger onset scale identified 39 patients who had anger episodes in the 2 hours before MI. The relative risk calculated for MI after anger was 2.3 (95% confidence interval). This was supported by the state anger subscale that was found to have a 1.9 (95% confidence interval) relative risk. However, use of aspirin and beta blockers reduced this risk. Thus the study authors concluded that episodes of anger can indeed trigger a heart attack, but that some medications may reduce the risk.

*SHEEP study.* Another case- crossover designed study that was conducted was the Stockholm Heart Epidemiology Program (SHEEP) that also assessed whether or not anger outbursts can trigger a nonfatal acute MI (Moller, Hallqvist, Diderichsen, Theorell, Reuterwall, & Ahlbom, 1999). This study included all cases of nonfatal, first MI's in Stockholm County, Sweden from April 1993 to December 1994. Participants' ages ranged from 45 to 70 years old. After exclusion of cases (missing data, unreliable information, fatality), 660 cases were left for analysis. Interviews were conducted by trained nurses while the patient was in the hospital or soon after patient discharge. The nurses used a training manual and individual instruction to maintain conformity. To control for recall bias, interviewers were not told about any hypothesis regarding the length of time between an anger episode and an MI. They were asked only to pay attention to the 26 hours preceding the cardiac event. Information was gathered on particulars such as time, type, circumstances, symptoms, and circumstances four days preceding the MI. The relative risk of MI during a 1- hour period after an intense anger episode was reported as 9.0(95% confidence interval). The trigger effect was increased for patients who reported their usual behavior as non-hostile. Use of aspirin and beta blockers were again observed to diminish the risk as in the Onset Study.

This study along with the Onset Study solidified psychological factors as potent triggers capable of affecting the heart, but the mechanisms by which this occurs is not clear. Several theories have been posited; these attempt to describe the pathophysiology between psychological factors and CHD.

*Pathophysiologic Theories*

There has been a dramatic increase in the 1990's of studies that examine psychological correlates of disease. A large-scale review of the field of psychoneuroimmunology examined studies published from 1939 to the present (Kiecolt-Glaser, McGuire, Robles, & Glaser, 2002). This review concluded that there is sufficient evidence that psychological factors interact with biological outcomes via the immune system. The studies clustered into themes such as stress, negative emotion, personality, and immune responses, and as a result pathophysiological theories have developed. Some of these pathways have received support and directed new hypothesis for future research. Key pathophysiologic theories that deal with CHD are elevated cardiovascular reactivity (CVR), inflammation, and platelet aggregation.

*Elevated cardiovascular reactivity (CVR).* Although investigations into anger and hostility have indicated a relationship to CHD (e.g., Fredrickson, Maynard, Helms, Haney, Siegler, & Barefoot, 2000; Houston, 1994; Suarez, Kuhn, Schanberg, Williams, & Zimmering, 1998; Suls & Wan, 1993), the pathophysiology is not clear. One of the hypothesized pathways leading to CHD that has been examined is sustained elevated cardiovascular reactivity (CVR) (Linden, Earle, Gerin, & Christenfield, 1997; Williams & Barefoot, 1988). The link is thought to be related to the repeated experience of the fight or flight response (Booth-Kewley & Friedman, 1987) through the experience of anger. Anger induces the body to release epinephrine (Suarez, Kuhn, Schanberg, Williams, & Zimmerman, 1998; Suarez, Shiller, & Kuhn, 1997), and vasodilatation

occurs in response to the epinephrine release in preparation for action (an adaptive function). This becomes problematic over time when the response occurs often and there is a lengthy resolution period to the previous non-aroused state (a maladaptive consequence of anger and hostility). Indeed, research has shown that the vascular spasms that occur due to anger reduce coronary vascular reserves (Boltwood, Taylor, Burke, Grogin, & Giacomini, 1993); thus, repeated exposure to the fight or flight response may become a health risk. In support of this hypothesis, a three-year prospective study (Markovitz, Matthews, Wing, Kuller, & Meilahn, 1991) measured anger and physical health in middle- aged women. This investigation found higher systolic and diastolic blood pressure in women who had higher Spielberger Trait Anger scores (Spielberger, Jacobs, Russell, & Crane, 1983) even when traditional risk factors were controlled.

Because activation of the fight or flight response is involuntary, examination of causal forces such as anger and hostility have been a logical target for intervention. In fact, in a random sampling of 1,478 coronary artery diseased patients, it was observed in controlled, long-term studies that those randomly chosen patients who received therapy aimed at altering hostile behavior (stress management), in addition to receiving traditional care demonstrated a reduction in myocardial re-infarction and mortality risk by approximately 50%, a decrease in myocardial ischemia, and an 8% decrease in coronary diameter (Blumenthal, et al., 1997; Frasure-Smith & Prince, 1989; Friedman, et al., 1986; Ornish, et al., 1998). A 28% increase in coronary diameter was observed in the controls who received standard medical care only. These studies highlight the need to

consider psychosocial aspects of an individual in addition to biological factors in optimal health care.

Another study (Friedman, Breall, Goodwin, Sparagon, Chandour, & Fleishman, 1996) found that patients who had CHD and silent ischemia diagnosed by Holter monitor reduced daily ischemic episodes by 50% following counseling for hostility and time urgency. Although it seems at face value to be a clear causal relation, there is nothing clear-cut about the connection. Indeed, the pathway to disease remains quite complex.

*Inflammation.* Atherosclerosis is often conceptualized as accumulation of plaque or fat within artery walls. When the accumulation becomes large enough, it can block blood flow possibly leading to CHD. However, atherosclerosis is not a passive circumstance. Atherosclerosis involves inflammation at every step of the disease process, because the endothelium (cell lining) does not function normally due to the inflammation process (Ross, 1999). The inflammation process may interrupt the endothelium of the artery and eventually lead to a rupture of the plaque. This can subsequently initiate an MI.

Vascular biology has demonstrated how C-reactive protein (a measure of inflammation) can predict CHD risk beyond traditional risk assessment. The Physicians' Health Study (Ridker, Cushman, Stampfer, Tracy, & Hennenkens, 1997) compared 543 subjects without MI or stroke with 543 subjects who developed CHD over the eight- year follow-up period. All subjects were randomized at the beginning of the study either into an aspirin group or a placebo group. The study found that the men who later had an MI

had significantly higher levels of C-reactive protein at baseline. The researchers concluded that the measurement of inflammation via C-reactive protein levels was a predictor of future MI independent of traditional risk factors.

Production of C-reactive protein has been found to increase with negative emotion. This is accomplished by emotion's effect on the immune system, which, in turn regulates the production of C-reactive protein. For instance, aggression was found to correlate significantly with T- and B- cell counts (immune function markers) in male military personnel regardless of age, health, or testosterone levels (Granger, Booth, & Johnson, 2000). Thus, CHD onset and progression is thought to be affected by negative emotion via the immune system's overproduction or under production of C-reactive protein.

*Platelet Aggregation.* Another pathophysiologic theory that has received support is platelet aggregation in response to anger. Wenneberg and colleagues (1997) found significant and positive correlations between anger expression and platelet aggregation. They posit the idea that exaggerated sympathetic nervous system response (fight or flight) results in a surge of catecholemines in the cardiovascular system, which encourages platelets to aggregate (get "sticky"). The platelet aggregation can lead to occlusive thrombus (blockage) formation in those who are susceptible.

A study by Markovitz (1998) supported this hypothesis when it was found that participants who displayed the greatest anger had increased platelet reactivity and hyperaggregability. This finding led the researchers to posit the further idea that it may

be the expression of anger that is the most health detrimental component, rather than the mere experience of anger.

This view is also held by Seigman (1993) who examined CHD in relation to anger suppression, anger expression, and anger experience. That investigation reported that it is the chronic expression of anger, not the experience or suppression of anger that was found to be a risk factor for CHD. The important term for consideration is "chronic" expression of anger, because this distinction alludes to a direction in research that has helped clarify state (temporary) from trait (enduring) anger and hostility.

Research continues to demonstrate the fact that to do a better job at preventing CHD, health care professionals must not rely solely on conventional risk factors. For instance, a study examining over 120,000 patients who suffered an acute MI found that 53% of women and 62% of men had 0 or 1 traditional risk factors (Khot, et. al, 2003). This leaves a great number of Americans at risk for CHD even if they know and assess conventional risk factors.

Although no definitive agreement on the biopsychosocial relatedness to CHD exists, enough evidence has been collected throughout the years to prompt the American Heart Association to add psychosocial evaluation and intervention into its Core Components of Cardiac Rehabilitation/Secondary Prevention program (AHA, 2000). This makes it all the more necessary to develop psychometrically sound measurements of these constructs such as anger, which is related to the primary goal of the present study.

## Research Hypothesis

1) The MAD-AS will be composed of several components of anger including (a) Anger Dyscontrol; (b) Angry Cognitions; (c) Verbal Expressions of Anger; (d) Physiological Arousal; (e) Anger Justification/Blame; (f) Externalization of Anger; and (g) Difficulty with Anger Resolution.
2) It is hypothesized that the MAD-AS total scale will significantly correlate with the STAXI-2 total scores, a well-established and widely used anger instrument in clinical adult medical outpatient sample.
3) The MAD-AS will demonstrate acceptable levels of alpha co-efficient reliability (between .70 and .90) for both total scores and also for subscale scores.
4) Subscale scores of the MAD-AS and STAXI-2 will significantly correlate.
5) The experimental cardiac group will demonstrate higher MAD-AS total scores and STAXI-2 total and trait anger scores than the non-cardiac control group.
6) Experimental cardiac group patients will demonstrate higher scores on the MAD-AS subscales than the non-cardiac control group.
7) The MAD-AS will demonstrate acceptable test-retest reliability (greater than .80).

# Chapter 2

# Methods

## *Participants*

Participants in the experimental group consisted of one hundred and fifty adult male cardiac outpatients between the ages of 35 and 63 who were recruited from a private cardiology practice. This age range was utilized because it is within the normed age range of the STAXI-2 (Spielberger, 1999) and it approaches the age range at which CHD is the predominant cardiovascular event (American Heart Association, 2004) in those with cardiac problems. Participants in the control group consisted of one hundred and fifty male non-cardiac patient adults recruited from a private dental practice; they were also between the ages of 35 - 63. The general demographic profile of the two geographic areas of the practices were found to be similar in total population, age, sex, racial composition, educational attainment, occupation, and SES according to the 2000 Census data.

## *Description of Measures*

*Demographic form.* A demographic form which was utilized in this study is provided in Appendix A. Questions were derived from the CHD risk factors established by the AHA and include smoking, overweight and obesity, physical inactivity,

hypertension, premature family history, high cholesterol, and diabetes. The directions requested that the participants complete the survey by checking the appropriate answer. Questions for the cardiac group began with the participant's age, sex, and marital status. Next followed questions about cardiac history: i.e., whether or not participants were diagnosed with a cardiac condition; whether or not they had a congenital condition ("have you had a heart condition since birth"); whether or not participants currently took medication for a cardiac condition and if so which ones they were taking (ACE inhibitors, calcium channel blockers, beta-blockers, diuretics, aspirin, or more than one medications). If the cardiac condition was congenital, the protocol was not utilized in data analysis because the likelihood that behavior factors were related to the heart defect was considered minimal. General medical history questions followed; these included participants' risk factors for heart disease: diabetes; high cholesterol (less than 200; 200 – 239; Above 240); whether or not participants smoked (less than a pack a day or more than a pack a day); whether or not participants exercised (moderate or vigorous); whether or not participants were overweight (10- 20 lbs; 20-30lbs; Over 30lbs); and whether or not patients had a family history of heart disease. Participants were also asked if they had hypertension and if so the medications they were taking to lower it. Medications to lower blood pressure may attenuate anger experience and/or expression. For example, these medications (beta blockers) are used for performers who have difficulty with stage fright (Davidson, 2003). Participants were then asked if they were currently in psychological counseling or currently taking antianxiety or antidepression medication, again possible factors than may impact anger experience and/or expression. Participants were finally

asked if they had any other major illness such as cancer or organ failure. Because the purpose of this study was to examine the anger/heart disease correlation, those protocols where these items were endorsed were not used in the data analysis.

The demographic sheet in the non-cardiac group differed only in the fact that participants were not asked about congenital heart illness, family history of heart disease, or heart medications because participants with a cardiac diagnosis were not utilized in the statistical analysis. As in the experimental cardiac group, those participants who endorsed the item indicating a serious medical condition such as cancer or organ failure were not utilized in this study. All other previously mentioned questions asked of the cardiac population were asked of the non-cardiac population.

*STAXI-2.* The State-Trait Anger Expression Inventory (STAXI-2) is a 57 item updated inventory (Spielberger, 1999) of the State-Trait Anger Expression Inventory (STAXI) (Spielberger, 1988). The STAXI-2 is a measure of the expression, experience, and control of anger. It was developed to evaluate anger's role in the development of physical illness such as heart disease, cancer, and hypertension. The inventory comprised six scales, five subscales, and an index of anger expression. Anger itself is conceptualized as having two major components, state and trait anger. State Anger (S-Ang) is conceptualized as a temporary psychobiological condition influenced by circumstance that varies from mild to intense. The S-Ang scale measures how intensely one experiences anger at the moment as well as the wish to express anger at the moment. The subscales of State Anger include Feeling Angry (A-Ang/F), measuring how intensely

a person is feeling angry at the moment; Feel Like Expressing Anger Verbally (S-Ang/V), measuring how intensely one wishes to express anger verbally at the moment; and Feel Like Expressing Anger Physically (S-Ang/P), measuring how intensely one wishes to express anger physically at the moment. Trait Anger is conceptualized as a more enduring mode of responding to situations in a similar manner, which includes heightened anger arousal. Individuals with high Trait Anger experience anger more often and with greater intensity than those with low Trait Anger. The Trait Anger (T-Ang) scales measures how often one experiences anger over a period of time. Subscales of T-Ang include Angry Temperament (T-Ang/T) that measures the proclivity to experience anger without provocation; and Angry Reaction (T-Ang/R) that measures how often angry feelings are experienced in frustrating situations or situations that involve negative evaluation. The Anger-Out Scale (AX-O) measures an individual's preference to exhibit the expression of anger either verbally or in a physically aggressive manner. Conversely, The Anger-In Scale (AX-I) measures how often angry feelings are repressed and not expressed. The Anger Control-Out Scale (AC-O) measures the attempt to control the expression of anger. The Anger Control-In (AC-I) measures how often one tries to gain control of the desire to express anger by gaining a sense of calmness following an angry episode. The Anger Expression Index (AX Index) is an overall index based on responses of AX-O, AX-I, AC-O, and AC-I.

The STAXI-2 is an easy to administer and easy to score instrument with strong psychometric properties. The STAXI-2 was reported to have a high internal consistency of .88 (Spielberger, 1999). Normative data for the measure is based on 1,900

hospitalized psychiatric patients (N = 276; 105 females, 171 males) and non-psychiatric patient adults (N = 1,644; 977 females, 667 males). The mean age of the normal adult sample was about 27 years old and the age range was 16 to 63. Psychological Assessment Resources (PAR) owns the copyright for the STAXI-2, thus all test materials (booklets, manual, answer sheets) were purchased through PAR.

*MAD-AS.* The Mahan and DiTomasso Anger Scale (MAD-AS), a 43- item anger scale (Mahan, 2001) that measures the cognitive, affective, and behavioral constructs of anger was developed in the quartet style of the Beck inventories (e.g. Beck, Epstein, Brown, & Steer, 1988). Thus, each item has four sentences that assess a certain aspect of anger and the participant rates each question on a four-point scale ranging from zero to three. Only items that received 100% agreement by experts in the field were chosen for this instrument.

The normative sample of the original MAD-AS investigation (Mahan, 2001) consisted of 3 groups of 60 participants, totaling 180 participants. Group 1 consisted of an inpatient psychiatric population (N = 60; 34 females, 26 males). Group 2 consisted of an outpatient psychiatric population (N = 60; 39 females, 21 males). Group 3 consisted of graduate students and nurses (N = 60; 50 females, 10 males).

Factor analysis of the scale suggests that it successfully measures cognitive, affective, and behavioral components of anger. High internal consistency for the entire scale was reported in the preceding three studies of the MAD-AS and were as follows: .96 (Mahan, 2001), .90 (Martin, 2002), and .94 (Beardmore, 2003). This scale is

advantageous because it is briefer, therefore easier to administer than other anger measurements. Permission to use the MAD-AS, obtained from Dr. DiTomasso and Dr. Mahan, is provided in Appendix B.

## Procedures

### *Experimental Group*

The experimental group of male cardiac outpatients was recruited from a local cardiology practice. A sign posted at the reception window indicated the physicians' participation in the research study; this is provided in Appendix A. When patients arrived for a scheduled appointment with one of the cardiologists, prospective participants who met the sex and age criteria were informed that they were eligible to take part in the research study by office staff. A trained office staff member approached those who expressed an interest in participation. Training of the office staff member included the use of a script that is provided in Appendix A. Office staff members who collected data were required to abide by all rules of confidentiality and to provide their signatures to attest to that fact (form provided in Appendix B). Eligible participants were told in general what the study was about (examination of the relation of emotions and health conditions), that study participation was voluntary, that they could choose not to participate or to discontinue participation at any time without consequence, and that all information would be kept anonymous (no identifying information was collected). The

participants were also informed that the Introduction/Participation Solicitation letter provided two local mental health referral telephone numbers in the unlikely event someone might become upset as a result of answering the questions. They were also provided with the telephone number of the principal investigators, Dr. Robert A. DiTomasso, Ph.D., A.B.P.P., and told they had permission to contact him. They were informed that they could contact the researcher via e-mail (provided on the form) if they desired an abstract detailing the results of the study.

If participants agreed to take part in the study they were given 1. the Introduction/Participation Solicitation letter, 2. the demographic sheet, 3. STAXI-2, and 4. MAD-AS. Signed letters of authorization for use of the MAD-AS, obtained from the authors, are provided in Appendix B. The STAXI-2 materials (provided in Appendix A) were purchased through Psychological Assessment Resources (PAR) who owns the copyright for the test. Participants were encouraged to complete the protocols before they left the office; however, those who were not able to complete the material and needed to take it home were given an addressed, postage paid, white envelope to return to the researcher via mail.

One third of the participants were given a retest packet in addition to the packet they were to fill out in the office. This packet contained the Introduction/Participation Solicitation letter (to reiterate confidentiality and voluntary status), the STAXI-2 and the MAD-AS and the participants were directed to fill out the materials one week later. The retest packets contained a $5 phone card as incentive to complete the forms and mail them back in the manila addressed, postage paid envelope as requested. The white and

manila colors were utilized as a color-coding method to differentiate the single test packets from the retest packets. The order in which the STAXI-2 and MAD-AS were presented in the packet was counterbalanced because in both groups and in all retest packets, half of the packets presented the STAXI-2 as the first instrument to complete whereas the other half were presented with the MAD-AS the first instrument to complete.

*Control Group*

The control group consisted of non-cardiac participants recruited from a private dental practice. The general demographic profile of the two geographic areas of the practices were found to be similar in total population, age, sex, racial composition, educational attainment, occupation, and SES, according to the 2000 Census data. A sign indicating notice of participation was posted at the reception window. A trained office staff member approached patients who were eligible to participate in the study. The office staff training and all other procedures were the same in both research groups.

When all the data was collected, the information was entered into an SPSS database Version 11.0 for Windows for statistical analysis. The data was independently entered and verified by the researcher.

*Research Design and Statistical Analysis*

To assess the psychometric properties of the MAD-AS, this study employed correlational research design; it also used between groups analysis where it was relevant.

The following psychometric analyses were conducted; 1) descriptive statistics on demographic data including means and standard deviations, 2) factor analysis with orothogonal varimax rotation of the MAD-AS, 3) Pearson R correlational analysis between the MAD-AS and STAXI-2 total scores, 4) co-efficient alpha reliability of the MAD-AS for total and subscales, 5) Pearson R correlational analysis of the MAD-AS and STAXI-2 subscale scores, 6) Multivariate analysis of variance (MANOVA) on the MAD-AS total scores and STAXI-2 total and trait anger scores by group, 7) MANOVA on MAD-AS subscale scores by group, 8) test-retest reliability coefficients computed by Pearson R correlational analysis. Significance level for this study was set at .05.

# Chapter 3

## Results

Several analyses were conducted to examine each of the seven research hypotheses utilizing a statistical significance level of .05. The descriptive statistics are reported first and include demographic information regarding age; subsequently, means and standard deviations are reported. Demographic information on marital status and CHD risk factors by group is then provided. To obtain a clear overall picture of the characteristics of each group and because previous research has found differences for various factors such as age (Spielberger, 1999), Chi Square tests were conducted on the demographic information to ascertain the presence of group differences. These differences are reported. A factor analysis with orothogonal varimax rotation was then conducted on the MAD-AS scores to determine which factors converged to create the subscales. To ascertain criterion validity of the MAD-AS, Pearson R correlational analysis between MAD-AS total scores and STAXI-2 total scores was conducted. To ascertain internal consistency of the MAD-AS, co-efficient alpha reliability of the total and subscales was conducted. To examine criterion validity of the MAD-AS further, Pearson R correlational analysis of the subscale scores of the MAD-AS and STAXI-2 was conducted. Then group differences in anger scores were examined by conducting multivariate analysis of variance (MANOVA) on the STAXI-2 and MAD-AS total scores and trait anger scores by group. To obtain more information on group differences in anger, a MANOVA on the MAD-AS subscale scores by group was conducted. Finally,

to ascertain test-retest reliability of the MAD-AS, test-retest reliability coefficients computed by Pearson R correlational analysis was conducted.

*Descriptive Statistics*

The Statistical Program for the Social Sciences, version 11.0 for Windows was utilized to create the database for this study. All data were independently entered and verified by the researcher. Verification consisted of the researcher's double- checking data entry for each response for every protocol and retest.

A total of 300 male participants between the ages of 35 - 63 chose to take part in this study, 150 in the experimental cardiac group and 150 in the non-cardiac control group. Professions in which the men worked included but were not limited to mechanics, welders, teachers, firefighters, managerial workers, attorneys, priests, retired persons, disabled persons, supervisors, and healthcare workers. Protocols were not used if the participant did not meet inclusion criteria for age, sex, and health status.

*Age.* The mean age for both groups combined was 50.35. The mean age was 47.49 for the non-cardiac group and 53.21 for the cardiac group. Table 1 provides statistics on means and standard deviation by group. Group age differences were observed, thus, a t test was conducted to confirm statistical significance. This test found significant differences ($t = 65.863, p < .001$).

Table 1

Age Statistics by Group

| | Non-Cardiac | Cardiac |
|---|---|---|
| Mean | 47.49 | 53.21 |
| SD | 8.45 | 8.23 |

*Marital status.* Of the participants in the non-cardiac group; one hundred and eighteen (78.7%) were married, sixteen (10.7%) were single, nine (6.0%) were divorced, four (2.7%) were cohabitating, three (2.0%) were separated, and none was widowed. Of the participants in the cardiac group; eleven (7.3%) were single, one hundred and twelve (74.7%) were married, five (3.3%) were separated, eighteen (12.0%) were divorced, two (1.3%) were widowed, and two (1.3%) were co-habitating. The majority of participants in both groups reported that they were married (non-cardiac = 78.7%; cardiac = 74.7%). The cardiac group differed because they reported double the divorce rate of the non-cardiac group (non-cardiac = 6% vs. cardiac = 12%); however, Chi Square analysis revealed that there was no statistical significance between the groups (Chi Square = 7.249, $p > .05$). A frequency distribution table of marital status by group is provided in Table 2.

Table 2

Frequency Distribution by Group: Marital Status

| | Non-Cardiac | | Cardiac | |
|---|---|---|---|---|
| | Frequency | Percent | Frequency | Percent |
| Single | 16 | 10.7% | 11 | 7.3% |
| Married | 118 | 78.7% | 112 | 74.7% |
| Separated | 3 | 2.0% | 5 | 3.3% |
| Divorced | 9 | 6.0% | 18 | 12.0% |
| Widowed | 0 | 0.0% | 2 | 1.3% |
| Co-Habitating | 4 | 2.7% | 2 | 1.3% |

*Risk factors in the non-cardiac group.* Of the participants in the non-cardiac control group, none had reported a cardiac diagnosis because those who endorsed this item were not used in the study. The exclusions were made to establish further the integrity of the control group because the study was examining those with confirmed heart disease in the experimental group in contrast to the control group who reported no heart disease. The majority, one hundred and thirty- eight (92.0%), reported that they did not have diabetes. Similarly, the majority, one hundred and eleven participants (75.0%) reported that they did not have hypertension. Three did not respond to this item. Of the thirty-six who reported that they did have hypertension, twenty-five were currently taking medication for the condition, eight were not, and three did not answer the question.

The majority of participants, one hundred and twelve (76.0%), reported that they did not have high cholesterol. Two did not respond to the item. Of those with high cholesterol, four (2.7%) reported a cholesterol level of less than 200, twenty-six (17.3%) reported cholesterol levels between 200 – 239, and eight (5.3%) reported cholesterol levels over 240.

One hundred and twenty participants (80.0%) reported that they did not smoke. Of those who did smoke, fifteen (10.0%) reported that they smoked less than a pack per day, and fifteen (10.0%) reported that they smoked more than a pack per day.

The majority of participants, ninety-six (64%), reported that they exercised. Of those who did exercise, fifty-one (34.0%) reported their exercise to be moderate and thirty-nine (26.0%) reported their exercise to be vigorous.

Eighty-one (54.0%) participants reported that they were overweight. Of those who reported they were overweight, forty-two (28.0%) reported they were less than 20lbs overweight, twenty-four (16.0%) reported they were 20 – 30 lbs overweight, and fifteen (10.0%) reported they were over 30 lbs overweight.

Only two, (1.3%), participants reported that they were currently in psychological counseling. Six (4.0%) reported that they were on antidepressants, five (3.3%) reported that they were on anti-anxiety medication.

*Risk factors in the cardiac group.* Of the participants in the cardiac group, all had a diagnosis of a heart condition because cardiac diagnosis was a criterion for inclusion in the study. As previously discussed, exclusion and inclusion criteria was set to establish the integrity of the groups because the study was examining those with confirmed heart disease in the experimental group in contrast to the control group who reported no heart disease. Of these cardiac patients, the majority, one hundred and fifteen (76.7%), reported that they were on multiple medications for the condition. Of those taking medications for a heart condition, fourteen (9.3%) were on aspirin therapy alone, nine (6.0%) were taking beta-blockers alone, two (1.3%) were taking Ace inhibitors alone, one (0.7%) was taking a calcium channel blocker alone, and one (0.7%) was on a diuretic alone.

One hundred and seventeen (78.0%) participants reported that they did not have diabetes. However, the majority in this cardiac group, eighty-three (55.3%), reported that

they did have hypertension. One did not respond to this item. Of those with hypertension, sixty-eight (45.3%) were taking medications and eight (5.3%) were not.

The majority of the cardiac group group, eighty-one (54.0%), reported that they did have high cholesterol. Five did not respond to this item. Of those with high cholesterol, forty-seven (31.3%) reported cholesterol levels under 200, twenty-six (17.3) reported cholesterol levels between 200 – 239, and thirteen (8.7%) reported cholesterol levels over 240.

One hundred and twenty seven (84.7%), the majority of the cardiac group, reported that they did not smoke. Of those who did smoke, one hundred and twenty-seven (84.7%) smoked less than a pack a day and twenty-three (15.3%) smoked more than a pack a day. Ninety-seven (64.7%) reported that they exercised. Of those who reported that they exercised, sixty-seven (44.7%) reported moderate activity, twenty-seven (18.0%) reported vigorous activity.

The majority in the cardiac group, ninety-nine (66.0%), reported to be overweight. One did not respond to this item. Of those who reported they were overweight, thirty-six (24%) were less than 20lbs overweight, twenty-eight (18.7%) were 20 – 30 lbs. overweight and thirty-four (22.7%) were more than 30lbs overweight.

Four (2.7%) participants in the cardiac group reported that they were currently receiving psychological counseling. Twenty-one (14.0%) reported they were on antidepressant medication, and twenty (13.3%) reported they were on anti-anxiety medication. The preceding information for both groups is provided in Table 3.

The majority of cardiac patients, ninety-seven (64.7%), reported that they had a family history of heart disease but forty-nine (32.7%) did not; four did not respond to this item.

*Group differences in risk factor profiles.* To obtain a clear overall picture of the health profile of each group, Chi Square tests were conducted on the risk factors to ascertain the presence of group differences. Risk factors that were found to have significant group differences were: diabetes (Chi Square = 11.529, $p < .001$), hypertension (Chi Square= 29.992, $p < .001$), cholesterol (Chi Square= 30.371, $p < .001$), total cholesterol level (Chi Square = 22.190, $p < .001$), amount of exercise (Chi Square = 4.266, $p < .05$), overweight (Chi Square = 4.546, $p < .05$), amount overweight (Chi Square = 6.581, $p < .05$), antidepressant medication use (Chi Square = 9.051, $p < .05$), and anti-anxiety medication use (Chi Square = 10.162, $p < .001$). These findings indicate that the cardiac group were individuals with significantly more health problems than the non-cardiac group.

Risk factors that did not reveal significant findings upon Chi Square analysis were: hypertension medication use (Chi Square = 3.456, $p > .05$), smoking (Chi Square = 1.123, $p > .05$), amount smoked (Chi Square = 2.342, $p > .05$), exercise (Chi Square = .040, $p > .05$), and psychological counseling (Chi Square = .694, $p > .05$). Table 4 provides this information.

Table 3
Frequency Distribution by Group: CHD Risk Factors

| | | No | Yes | Supplemental Info |
|---|---|---|---|---|
| **Non-Cardiac** | Diabetes | 92.0% | 8.0% | |
| | Hypertension | 74.0% | 24.0% | Meds Yes = 16.7%<br>Meds No = 5.3% |
| | High Cholesterol | 74.7% | 24.0% | <200 = 2.7%<br>200-239 = 17.3%<br>>240 = 5.3% |
| | Smoking | 80.0% | 20.0% | < Pack/Day = 10.0%<br>> Pack/Day = 10.0% |
| | Exercise | 36.0% | 64.0% | Moderate = 34.0%<br>Vigorous = 26.0% |
| | Overweight | 45.3% | 54.0% | < 20lbs = 28.0%<br>20-30lbs = 16.0%<br>>30 = 10.0% |
| | Counseling | 97.3% | 1.3% | |
| | Antidepressant | 95.3% | 4.0% | |
| | Anti-anxiety | 96.7% | 3.3% | |
| **Cardiac** | Diabetes | 78.0% | 22.0% | |
| | Hypertension | 44.0% | 55.3% | Meds Yes = 45.3%<br>Meds No = 5.3% |
| | High Cholesterol | 42.7% | 54.0% | <200 = 31.3%<br>200-239 = 17.3%<br>>240 = 8.7% |
| | Smoking | 84.7% | 15.3% | < Pack/Day = 10.0%<br>> Pack/Day = 4.0% |
| | Exercise | 34.7% | 64.7% | Moderate = 44.7%<br>Vigorous = 18.0% |
| | Overweight | 33.3% | 66.0% | <20lbs = 24.0%<br>20-30lbs = 18.7%<br>> 30lbs = 22.7% |
| | Counseling | 95.3% | 2.7% | |
| | Antidepressant | 86.0% | 14.0% | |
| | Anti-anxiety | 84.7% | 13.3% | |

Table 4

Significant Group Differences: Risk Factors

| RISK FACTOR | CHI SQUARE |
|---|---|
| Diabetes | 11.529** |
| Hypertension | 29.992** |
| Hypertension Medication Use | 3.456 |
| High Cholesterol | 30.371** |
| Total Cholesterol Level | 22.190** |
| Smoking | 1.123 |
| Amount Smoked | 2.342 |
| Exercise | .040 |
| Amount of Exercise | 4.266* |
| Overweight | 4.546* |
| Amount Overweight | 6.581* |
| Psychological Counseling | .694 |
| Antidepressant Medication Use | 9.051* |
| Anti-anxiety Medication Use | 10.162** |

* Significant at the $p < .05$ level
** Significant at the $p < .001$ level

*Factor Analysis of the MAD-AS*

A principal component, varimax rotated factor analysis using Eigenvalues greater than 1 was conducted. Six factors accounting for 51.08% of the variance were extracted. The factor loading criterion of $\geq .45$ was utilized to determine which items were retained on a given factor (see Table 6).

Factor 1, Anger Resolution, comprised five items. These items measured difficulty getting over angry feelings "I have trouble letting go of things that have angered me in the past", a need to get even, feeling bitter, experiencing thoughts of hurting others when angry, and holding grudges. Those who are high in this factor dimension have difficulty resuming baseline anger levels after an angry episode.

Factor 2, Verbal Expression, was comprised of nine items. Items include a tendency to be critical and argumentative "When people disagree with me, I argue" "I am critical of others when angry", proneness to place blame on others for anger, overtly showing displeasure, and retaliatory behavior. Those who score high on this factor have a need to show and express anger and believe it is due to other external factors.

Factor 3, Behavioral Dyscontrol, contained seven items. Those who score high in this factor have a tendency to experience anger more frequently "I anger more frequently than most people" often without reason "I get angry without reason", they throw objects when angry, they feel that they do not have control over anger, and they do not get over anger quickly. Those who score high on this subscale overtly display their anger, are

easily provoked to anger, and can become angry without a particular circumstance to precipitate the outburst.

Factor 4, Physical Aggression, consisted of four items. These items tapped into the tendency to feel others intend to cause them anger "People intend to anger me" and they physically lash out at others "When provoked, I hit people". Those high on this scale tend to be more violent when angry.

Factor 5, Physiological Arousal, contained 4 items. This scale captured the physiologic dimension of anger, which includes feelings of restlessness "When angry, I feel restless or agitated", muscle tension, rapid breathing, and rapid heart rate. Those high on this scale are more aware of the physical sensations related to the emotion of anger.

Factor 6, Anger Justification, contained 3 items. These items describe the tendency to ruminate and let anger interfere with sleep "My anger keeps me up at night", and attribute anger to situational circumstances "When under stress, I get angry"; "In difficult situations, I get angry". Those who score high on this subscale have a tendency to expend a great deal of energy justifying explanatory causes of their anger.

Descriptive statistics including means and standard deviations of the six subscales are provided in Table 5.

Table 5

Subscale Descriptive Statistics

| | MEAN | STANDARD DEVIATION |
|---|---|---|
| F1: Anger Resolution | 4.30 | 2.82 |
| F2: Verbal Expression | 7.80 | 3.67 |
| F3: Behavioral Dyscontrol | 4.83 | 3.53 |
| F4: Physical Aggression | 1.11 | 1.43 |
| F5: Physiological Arousal | 3.85 | 2.57 |
| F6: Anger Justification | 2.16 | 1.38 |

Table 6

MAD-AS Factor Loadings of the Principle Components Varimax Rotated Analysis

*Factor 1: Anger Resolution*

*Alpha = .82 Eigenvalue = 12.90 Variance = 29.99% Cumulative Variance = 29.99%*

| Number | Item | Factor Loading |
|---|---|---|
| 7. | I have trouble letting go of things that have angered me in the past | .747 |
| 8. | I hold grudges against those who have angered me | .734 |
| 3. | I have trouble letting go of my anger | .620 |
| 1. | I feel a need to get even with those who anger me | .607 |
| 33. | I feel bitter about things | .509 |
| 19. | When I am angry I have thoughts of hurting others | .467 |

*Factor 2: Verbal Expression*

*Alpha = .84 Eigenvalue = 2.23 Variance = 5.18% Cumulative Variance = 35.18%*

| Number | Item | Factor Loading |
|---|---|---|
| 27. | When angry, I let it show | .647 |
| 30. | I am argumentative | .594 |
| 23. | The behavior of others causes me to get angry | .529 |
| 42. | When someone offends me I retaliate | .527 |
| 14. | I am critical of others when angry | .514 |

| | |
|---|---|
| 16. I blame others for my anger | .484 |
| 26. I insult people when I am angry | .477 |
| 32. When people disagree with me, I argue | .472 |

*Factor 3: Behavioral Dyscontrol*

*Alpha = .74 Eigenvalue = 2.06 Variance = 4.79% Cumulative Variance = 39.96%*

| Number | Item | Factor Loading |
|---|---|---|
| 11. | I cannot control my anger | .740 |
| 5. | I get angry without reason | .567 |
| 28. | I lose control when angry | .559 |
| 6. | I am quick to anger | .519 |
| 36. | Once angry, I never get over it quickly | .483 |
| 10. | I throw things when I am angry | .472 |
| 4. | I anger more frequently than most people | .449 |

*Factor 4: Physical Aggression*

*Alpha = .72 Eigenvalue = 1.80 Variance = 4.20% Cumulative Variance = 44.16%*

| Number | Item | Factor Loading |
|---|---|---|
| 12. | I hit those who anger me | .797 |
| 34. | When provoked, I hit people | .766 |
| 20. | People intend to anger me | .582 |
| 29. | I threaten people when angry | .545 |

*Factor 5: Physiological Arousal*

*Alpha = .84 Eigenvalue = 1.53 Variance = 3.55% Cumulative Variance = 47.71%*

| Number | Item | Factor Loading |
|---|---|---|
| 38. | When angry, I feel my heart beating faster | .801 |
| 40. | When angry, my breathing is rapid | .761 |
| 39. | When angry, my muscles feel tense | .752 |
| 41. | When angry, I feel restless or agitated | .665 |

*Factor 6: Anger Justification*

*Alpha = .68 Eigenvalue = 1.45 Variance = 3.37% Cumulative Variance = 51.08%*

| Number | Item | Factor Loading |
|---|---|---|
| 43. | In difficult situations, I get angry | .604 |
| 35. | When under stress, I get angry | .465 |
| 2. | My anger keeps me up at night | .450 |

*Correlations Between the MAD-AS and STAXI-2 Total Scores*

Correlational analysis was conducted between the MAD-AS and STAXI-2 total scores and was found to be statistically significant. The Pearson Product Moment Correlation between total scores for the MAD-AS and STAXI-2 scales was .274 ($p < .01$) and is provided in Table 7.

Table 7.

Correlation between MAD-AS and STAXI-2 Total Scores

| | | STAXI –2 TOTAL |
|---|---|---|
| MAD-AS TOTAL | Pearson Correlation | .274** |
| | Sig. (1-tailed) | .000 |
| | N | .255 |

** Correlation is significant at the $p < .01$ level (1-tailed).

*Coefficient Alpha Reliability of the MAD-AS Total and Subscale Scores*

Internal consistency for the MAD-AS total scores and subscale scores was conducted using Chronbach's coefficient alpha reliability. Coefficient alpha for the entire scale was .93. Coefficient alpha for the subscale scores were as follows: Factor 1, .82; Factor 2, .84; Factor 3, .74; Factor 4, .72; Factor 5, .84; Factor 6, .68. Inter-correlations of the MAD-AS subscales are provided in Table 8.

Table 8

Pearson Inter-Correlations of the MAD-AS Factors

| | Factor 1 | Factor 2 | Factor 3 | Factor 4 | Factor 5 | Factor 6 |
|---|---|---|---|---|---|---|
| Factor 1 | -- | .667** | .619** | .439** | .461** | .541** |
| Factor 2 | | | .684** | .497** | .504** | .579** |
| Factor 3 | | | | .479** | .458** | .581** |
| Factor 4 | | | | | .365** | .335** |
| Factor 5 | | | | | | .493** |
| Factor 6 | | | | | | |

Note: Significant at $p < .001$ level (one-tailed)

*Correlations Between the MAD-AS and STAXI-2 Subscale Scores*

Pearson R Product Moment Correlational Analysis was conducted between the MAD-AS and STAXI subscales to examine the relationship between the scores. Correlations ranged from -.202 to .748 and were all significantly correlated with the exception F5 Physiological Arousal and SANGP State Anger- Physical. Table 9 provides the inter-correlations of these subscales.

Table 9
Pearson Inter-Correlations Between MAD-AS and STAXI-2 Subscales

STAXI-2 Subscales MAD-AS Subscales

| | | F1ANGRES | F2VERBAL | F3BEHDYS | F4PHYAGG | F5AROUS | F6ANGJUS |
|---|---|---|---|---|---|---|---|
| STATE | Pearson Correlation | .291** | .263** | .293** | .219** | .138** | .352** |
| | Sig. (1-tailed) | .000 | .000 | .000 | .000 | .009 | .000 |
| | N | 294 | 291 | 294 | 292 | 295 | 294 |
| TRAIT | Pearson Correlation | .631** | .703** | .586** | .596** | .462** | .491** |
| | Sig. (1-tailed) | .000 | .000 | .000 | .000 | .000 | .000 |
| | N | 295 | 292 | 295 | 295 | 296 | 295 |
| SANGF | Pearson Correlation | .241** | .258** | .286** | .197** | .164** | .318** |
| | Sig. (1-tailed) | .000 | .000 | .000 | .000 | .002 | .000 |
| | N | 297 | 294 | 297 | 295 | 298 | 297 |
| SANGV | Pearson Correlation | .257** | .221** | .260** | .198** | .117** | .316** |
| | Sig. (1-tailed) | .000 | .000 | .000 | .000 | .022 | .000 |
| | N | 298 | 295 | 298 | 296 | 299 | 298 |
| SANGP | Pearson Correlation | .285** | .206** | .223** | .194** | .080 | .283** |
| | Sig. (1-tailed) | .000 | .000 | .000 | .000 | .083 | .000 |
| | N | 297 | 294 | 297 | 295 | 298 | 297 |
| TANGT | Pearson Correlation | .513** | .646** | .748** | .363** | .402** | .476** |
| | Sig. (1-tailed) | .000 | .000 | .000 | .000 | .000 | .000 |
| | N | 297 | 294 | 297 | 295 | 298 | 297 |
| TANGR | Pearson Correlation | .470** | .578** | .425** | .216** | .370** | .395** |
| | Sig. (1-tailed) | .000 | .000 | .000 | .000 | .000 | .000 |
| | N | 297 | 295 | 297 | 295 | 298 | 297 |
| AXO | Pearson Correlation | .475** | .600** | .579** | .445** | .355** | .339** |
| | Sig. (1-tailed) | .000 | .000 | .000 | .000 | .000 | .000 |
| | N | 295 | 292 | 295 | 293 | 296 | 295 |
| AXI | Pearson Correlation | .580** | .452** | .422** | .275** | .409** | .434** |
| | Sig. (1-tailed) | .000 | .000 | .000 | .000 | .000 | .000 |
| | N | 287 | 285 | 287 | 285 | 288 | 287 |
| ACO | Pearson Correlation | -.411** | -.476** | -.640** | -.392** | -.272** | -.377** |
| | Sig. (1-tailed) | .000 | .000 | .000 | .000 | .000 | .000 |
| | N | 291 | 288 | 291 | 289 | 292 | 291 |
| ACI | Pearson Correlation | -.410** | -.423** | -.534** | -.367** | -.202** | -.344** |
| | Sig. (1-tailed) | .000 | .000 | .000 | .000 | .000 | .000 |
| | N | 292 | 290 | 292 | 290 | 293 | 292 |
| AXIIND | Pearson Correlation | .604** | .625** | .723** | .484** | .396** | .488** |
| | Sig. (1-tailed | .000 | .000 | .000 | .000 | .000 | .000 |
| | N | 273 | 271 | 273 | 271 | 274 | 273 |

** Correlations Significant at $p < .001$ (1-tailed)

*Group Differences between MAD-AS Total Scores and STAXI-2 Total and Trait Anger Scores*

A multivariate analysis of variance (MANOVA) was conducted on the MAD-AS total scores and STAXI-2 total and trait anger scores to determine if there were significant differences between the cardiac and non-cardiac groups. The research hypothesis was that scores would be higher in total anger scores on the MAD-AS and STAXI-2 and trait anger on the STAXI-2 in particular. This test revealed non-significant findings, Wilks Lambda = .988, F (3, 251) = 1.002, $p > .05$, indicating no overall differences between the groups. However, based on detection of age effects in anger (Spielberger, 1999), a MANCOVA using age as a covariate was conducted. This test revealed significant findings, Wilks Lambda = .965, F (3,250) = 3.034, $p < .05$. It was found that the MAD-AS total scores were significant, F = 6.427, $p < .05$, and STAXI-2 total scores, F = 1.225, $p > .05$ and STAXI-2 trait anger scores F = 1.348, $p > .05$ were not significant.

The findings regarding age initiated the prospect that the MAD-AS may also be proficient enough to detect differences if significant demographic variables were controlled for. To test this hypothesis, the significant variables had to be established by conducting Chi Square analysis on reported demographic factors. Significant factors that were identified were hypertension, diabetes, overweight, cholesterol, antidepressant use, and anti-anxiety medication use. A MANCOVA was conducted using these factors as covariates. The underlying assumption was that if these factors were controlled for,

group differences might be detected. This test was found to be significant, Wilks Lambda = .926, F (2, 83) = 3.301, $p < .05$. Hence, when hypertension, diabetes, overweight, cholesterol, antidepressant use, and anti-anxiety medication use were controlled for, the cardiac group had significantly higher levels of anger than the non-cardiac group as measured by the MAD-AS, F = 6.656, $p < .05$, but not for the STAXI-2, F = .050, $p > .05$.

*Group Differences between MAD-AS Subscale Scores*

A multivariate analysis of variance (MANOVA) was conducted on the MAD-AS subscale scores in the cardiac and non-cardiac groups to determine if there were differences. The research hypothesis was that the cardiac group would score higher on the MAD-AS subscales than would the non-cardiac group. This test revealed non-significant results, Wilks Lambda = .968, F (6, 283) = 1.545, $p > .05$. Again, because age effects in anger have been reported (Spielberger, 1999), a multivariate analysis of covariance (MANCOVA) was conducted using age as a covariate. This test was significant, Wilks Lambda = .943, F (6,282) = 2.839, $p < .05$. Significant subscales that were observed were: F2 Verbal Expression F = 3.942; $p < .05$; F3 Behavioral Dyscontrol F = 6.021, $p < .05$; and F4 Physical Aggression F = 10.188, $p < .05$.

*Test-Retest Reliability of MAD-AS*

Test-retest reliability refers to an instrument's stability over time; therefore, scores from one administration of a measure at a particular point in time should significantly correlate with scores from the same instrument at another point in time if the measure is reliable. This study used a one- week period of elapsed time between administrations from the first period to the retest period. Of the 300 participants in the study, one-third (100) were given a retest. Sixty-five participants completed and returned the retests. MAD-AS total score test-retest reliability coefficients was computed using Pearson R correlational analysis was .87 ($p < .01$, one-tailed) and is provided in Table 10.

Table 10

Test-Retest Correlational Analysis

| | | RMADASTOT |
|---|---|---|
| MADASTOT | Pearson Correlation | .872** |
| | Sig. (1-tailed) | .000 |
| | N | 62 |

** Correlation is significant at the $p < .01$ level (1-tailed).

# Chapter 4

# Discussion

This is the fourth study that examined the MAD-AS as a psychometrically sound instrument measuring self-reported anger; it the first study to do so in an outpatient cardiac population. The first study (Mahan, 2001) found strong psychometric properties that appear to measure successfully the cognitive, physical, and behavioral aspects of anger. The second study (Martin, 2002) found a similar factor structure and introduced a Significant Other (SO MAD-AS) version of the instrument. The third study (Beardmore, 2003) again observed a similar and significant factor structure of the MAD-AS in an outpatient population. The present study is able to lend further credibility to the psychometric soundness of the MAD-AS by observing again a similar factor structure, as well as strong reliability and validity. This research makes an important contribution to the study of anger because the MAD-AS is a proving to be a robust anger measure that takes less time than other measures. As such, the original goal of the Mahan (2001) study, which was to introduce the MAD-AS as a screening tool similar to the Beck inventories, may be realized.

## *Descriptive Statistics*

*Age statistics.* Normative data for the STAXI-2 is based on 1,900 hospitalized psychiatric patients (N = 276; 105 females, 171 males) and non-psychiatric patient adults

(N = 1,644; 977 females, 667 males). The mean age of the normal adult sample is approximately 27 years and the age range is from 16 to 63. Of the 1,900 normed STAXI-2 subjects, 838 were males. Of that male sample, 667 were normal adults as contrasted to psychiatric patients. Of the 667 non-psychiatric males, only129 were 30 years old and older. Hence, for the purposes of the current study, the comparison group from the STAXI-2 is composed of 129 subjects. The current study looked specifically at 300 male participants in the 35-63 year old age range, which is a significant issue given that the average age for a male's having a first MI is 65.8 (AHA, 2004). Indeed, the current study found statistically significant differences because the non-cardiac group was composed of younger, healthier men and the men in the cardiac group were older less healthy.

*Risk factors.* A significant difference was observed between the cardiac group and the non-cardiac group regarding risk factors. An examination of health profiles denotes two groups with distinct characteristics. The cardiac group reported a significantly higher rate of diabetes, hypertension, high cholesterol, and overweight status as compared with the non-cardiac group. Thus, the cardiac group demonstrated the presence of many risk factors related to CHD. This supports the fact that conventional risk factors are highly associated with CHD and must be modified to benefit health status. However, it was an interesting finding that hypertension medication use between both groups was not significant. This implies the fact that of those participants who have hypertension, similar ratios take medications to control it regardless of group status.

Smoking status and amount smoked was not significantly different between groups. In fact, more participants in the non-cardiac group smoked, and smoked greater amounts (greater than 1 pack per day). This is a finding that makes sense if one considers the health status circumstances of each group. The non-cardiac group comprised younger men who do not have any significant medical diagnosis; therefore, they are exposed to the same amount of anti-smoking messages as most Americans via media, family, and friends. As such, the majority of the group does not smoke; however, those who do smoke have not had any harsh health consequence from the habit to strongly dissuade tobacco use. On the other hand, the cardiac group is in treatment for a major medical condition that is directly related to tobacco use. There is a great likelihood that these participants have been told by several healthcare providers that they must stop smoking because it has contributed to their CHD. They most likely have family members that have done their best to help improve overall health habits and discourage smoking. These participants had a rather substantial scare and many have taken the scare seriously and accordingly have abstained from tobacco.

Another finding that is of note is that exercise habits were not significant between the groups; however, the amount of exercise in which one engaged was significant. The fact that over 64% of all participants reported that they got exercise is encouraging given that approximately 55% of Americans do not exercise (AHA, 2004). Again, these findings logically follow if one considers the circumstances of the participants. For instance, more cardiac participants may be exercising now because of the CHD diagnosis and promptings by healthcare professionals. They may be involved in cardiac

rehabilitation, a supervised exercise program particularly designed for cardiac patients. This would coincide with the finding that the cardiac group exercised more moderately (activities such as walking or bicycling lasting at least 30 minutes on 5 or more of 7 days) as opposed to more vigorously (activity causing hard breathing and sweating for at least 20 minutes on 3 or more of 7 days). Conversely more non-cardiac participants exercised more vigorously than moderately. Age may also play a role in amount of exercise because the non-cardiac group is a younger group. Also, if many exercisers in the cardiac group did not get exercise before their diagnosis but are doing so now in response to a health threat, they may be more apt to do the minimum (moderate) amount required. An older man in his 50's who begins an exercise program for the first time in many years will most likely lack the exercise capacity of a younger man in his 30's who has been exercising throughout his life.

Fear may also play a role in cardiac patients' willingness to engage in vigorous exertion. Many of the physiological sensations that can be a result of vigorous exercise (sweating, lightheadedness, chest discomfort) are similar to the symptoms of a heart attack. It may be difficult for many of the cardiac participants to push themselves to the vigorous level for fear that pushing too far may initiate an MI. Certainly, a similar thought process plays a role in the return to sexual intimacy (Moser & Dracup, 1995) because the patient does not return to pre-MI sexual frequency (Hamilton & Seidman).

It was found that the cardiac group reported significantly more antidepressant and anti-anxiety medication use. It is logical to surmise that dealing with a major medical condition places enough stressors on one to initiate the use of medication. More contact

with medical professionals may also give rise to more opportunity to discuss problems, thus, the cardiac group may simply have had more occasion to address overall functioning than the non-cardiac group. In our managed care environment, addressing only significant medical issues is more of the norm than a long discussion of the patient's mental state in an office visit. It could also be argued that those who have psychological issues prompting one to engage in unhealthy behaviors that result in CHD are more likely to use psychotropic medication. This "catch-22"has been an issue in the assessment of psychological correlates of CHD, resulting in inconsistent findings.

*Construct Validity*

Construct validity is the extent to which an instrument accurately measures a particular construct (Kazdin, 1992), in this case anger. The factor analysis of the MAD-AS in the present study supports its construct validity, following a similar pattern as previous research examining the instrument (Beardmore, 2003, Mahan, 2001, & Martin, 2002). The factor analysis in this study revealed 6 subscales: anger resolution, verbal expression, behavioral dyscontrol, physical aggression, physiological arousal, and anger justification. The original Mahan study (2001) identified seven subscales: anger dyscontrol, angry cognitions, verbal expressions of anger, physiological arousal, anger justification/blame, externalization of anger, and difficulty with anger resolution. The Martin (2002) study identified six subscales: difficulty with anger resolution, emotional dycontrol, physiological arousal, physical anger/aggression, argumentativeness, and

display of anger. Finally, Beardmore (2003) found six subscales: behavioral dyscontrol, anger resolution, aggression, physiological arousal, externalization of anger, and verbal expression of anger.

All four studies contain a physiological arousal subscale and an anger resolution subscale. All four studies contain a subscale consisting of anger expressed vocally: Mahan - Verbal expressions of anger; Martin -Argumentativeness; Beardmore - Verbal expressions of anger; Current study - Verbal expression. All four studies found a subscale that addresses anger out of control: Mahan – Behavioral dyscontrol; Martin – Emotional dyscontrol; Beardmore – Behavioral dyscontrol; Current study – Behavioral dyscontrol. All studies found a subscale that addresses physical aggression: Mahan – Behavioral dyscontrol; Martin – Physical anger/aggression; Beardmore – Aggression; Current study – Physical aggression. Three of the four studies found a scale that is composed of outward blame for anger: Mahan – Anger justification/blame; Beardmore – Externalization of anger; Current study – Anger justification. Hence, it is clear that all four MAD-AS studies have followed a similar factor structure.

Construct validity also refers to the extent to which an instrument measures what it claims to measure, in this case anger. One way to establish construct validity is to measure its relatedness to another established measure of the same construct (Gravetter & Wallnau, 2000). In this study, the STAXI-2 was used to establish validity by comparing both total scores as well as subscale scores of the measure with the MAD-AS.

*Correlations between the MAD-AS and STAXI-2 total scores.* The MAD-AS total scores significantly correlated with the STAXI-2 total scores. The Pearson Product Moment Correlation between total scores for the scales was .274, $p < .01$. However, because the correlation did not appear to be very strong, and age appeared to be a factor in scores, a partial correlation was conducted. Partial correlations involve partialling out, or controlling for, the effects of one or more variables (Howell, 1997). Age was utilized in this case as the variable controlled for. This test found a slightly higher correlation (.284, $p < .01$) but it was not dramatically higher. Thus, the correlation between MAD-AS and STAXI-2 total scores moderately support the construct validity of the MAD-AS.

*Correlations between the MAD-AS and STAXI-2 subscales.* Another measure of construct validity was the comparison of MAD-AS subscales with the STAXI-2 subscales. All subscales of the MAD-AS and STAXI-2 had significant inter-correlations with the exception of F5 Physiological Arousal and S/ Ang-P (state anger- physical). The fact that these two subscales did not correlate makes logical sense, given the fact that an individual who is not angry at the moment would not experience any physiological arousal. There were several strong correlations that were observed such as: T-Ang and F2 verbal; T-Ang and F1 anger resolution; T-Ang/T and F2 verbal; T-Ang/T and F3 behavioral dyscontrol; and AX-O and F2 verbal expression.

Trait anger is conceptualized as a more enduring mode of responding to situations in a similar manner, which includes heightened anger arousal. Individuals with high trait anger experience anger more often and with greater intensity than those with low trait

anger. The trait anger (T-Ang) scales measure how often one experiences anger over a period of time, thus the high correlation with the MAD-AS F2: verbal expression subscale is logical, given that the verbal subscale includes items that indicate an individual's propensity to be critical of others, to let anger show, to be insulting and argumentative and to believe that is others who are at fault for his or her anger.

STAXI-2 trait anger and MAD-AS F1 anger resolution significantly correlate, which is also a logical connection. Again, trait anger is conceptualized as a more enduring mode of responding to situations with heightened anger arousal. Thus, if someone experiences frequent and intense anger and has the propensity to attribute his or her anger to other's behavior (as correlated with F2 verbal expression), a significant correlation with F1 anger resolution would be consistent. F1 anger resolution deals with one's difficulty in letting go of anger, in holding grudges, in feeling a need to get even, and in feeling a desire to physically harm others when angry. Thus, if individuals often and intensely experiences anger that they believe is due to other people's behavior, they would have difficulty letting it go and returning to pre-anger status. They have felt the effects of something they do not believe is under their control so they are quite angry about it and are not so willing to let it go.

STAXI-2 trait anger – temperament (T-Ang/T) measure one's tendency to become angry without provocation. This subscale significantly correlated with the MAD-AS F2 verbal expression subscale. As previously discussed, the F2 verbal expression subscale indicates an individual's propensity to be critical of others, to let anger show, to be insulting and argumentative and to believe that is others who are at

fault for his or her anger. These two scales correlate because an individual who has the tendency to fly off the handle without being provoked would be liable have the tendency to blame others for the occurrence because it would help rationalize the anger reaction.

Once more, the STAXI-2 trait anger – temperament (T-Ang/T) subscale measures one's tendency to become angry without provocation. The MAD-AS F3 behavioral dyscontrol subscale measures one's propensity to be quick to anger frequently without reason and to throw things and feel a loss of control. It is reasonable to deduce that these subscales highly correlate for the reason that they both deal with intense, frequent, unprovoked periods of intense anger.

The STAXI-2 anger expression out subscale (AX-O) measures the preference to exhibit the expression of anger in a verbally or physically aggressive manner. As previously mentioned, the MAD-AS F2 verbal expression subscale indicates an individual's propensity to be critical of others, to let anger show, to be insulting and argumentative and to believe that is others who are at fault for his or her anger. These scales correlate because they both address one's verbal and/or physical aggressiveness in anger expression.

*Reliability*

Reliability generally refers to the consistency of a measure, which is an instrument's ability to produce stable, consistent measurements (Gravetter & Wallnau, 2000). To assess the reliability of the MAD-AS, the internal consistency as measured by

Chronbach's alpha and test-retest reliability as measured by Pearson R correlational analysis was calculated.

*Internal consistency.* Internal consistency for the MAD-AS total scores and subscale scores was conducted using Chronbach's coefficient alpha reliability. Coefficient alpha for the entire scale was found to be a robust .93. Coefficient alpha for the MAD-AS subscale scores were also strong and are as follows: Factor 1, .82; Factor 2, .84; Factor 3, .74; Factor 4, .72; Factor 5, .84; Factor 6, .68. Thus, this study supports the notion that the MAD-AS does, in fact, measure the anger constructs that it was developed to measure.

*Test-retest reliability of MAD-AS.* Test-retest reliability refers to an instrument's stability over time; therefore, scores from one administration of a measure at a particular point in time should significantly correlate with scores from the same instrument at another point in time if the measure is reliable. This speaks to the measure's consistency. This study used a one- week period of elapsed time between administrations. Of the 300 participants in the study, one-third (100) were given a retest. Sixty-five participants completed and returned the retests. MAD-AS total score test-retest reliability coefficients were computed using Pearson R correlational analysis. Test retest reliability was found to be .87, demonstrating strong temporal stability. For comparison sake, the STAXI-2 reported test-retest reliability as .88.

*Group Differences Between MAD-AS Total Scores and STAXI-2 Total and Trait Anger Scores*

A multivariate analysis of variance (MANOVA) was conducted on the MAD-AS total scores and STAXI-2 total and trait anger scores to determine if there were significant differences between the cardiac and non-cardiac groups. The research hypothesis was that scores would be higher in total anger scores on the MAD-AS and STAXI-2 and trait anger on the STAXI-2 trait anger subscale in particular. This test revealed non-significant findings, Wilks Lambda = .988, F (3, 251) = 1.002, $p > .05$, indicating no overall differences between the groups. However, based on detection of age effects in anger (Spielberger, 1999), a MANCOVA using age as a covariate was conducted. This test revealed significant findings, Wilks Lambda = .965, F (3,250) = 3.034, $p < .05$. It was found that the MAD-AS total scores were significant, F = 6.427, $p < .05$, and the STAXI-2 total scores, F = 1.225, $p > .05$ and STAXI-2 trait anger scores F = 1.348, $p > .05$ were not significant. This finding is unexpected because the MAD-AS appears to be uncovering some aspect of anger masked by age.

The findings regarding age initiated the prospect that the MAD-AS may also be proficient enough to detect differences if significant demographic variables were controlled for. To test this hypothesis, the significant variables were derived by conducting Chi Square analysis on reported demographic factors. Significant factors that were identified were hypertension, diabetes, overweight, cholesterol, antidepressant use, and anti-anxiety medication use. A MANCOVA was conducted using these factors

as covariates. The underlying assumption was that if these factors were controlled for, group differences might be detected. This test was found to be significant Wilks Lambda = .926, F (2, 83) = 3.301, $p < .05$. Hence, when hypertension, diabetes, overweight, cholesterol, antidepressant use, and anti-anxiety medication use were controlled for, the cardiac group had significantly higher levels of anger than the non-cardiac group as measured by the MAD-AS, F = 6.656, $p < .05$, but not the STAXI-2, F = .050, $p > .05$. These findings support the concept that anger may indeed play a role in CHD, highlighting the notion that future investigations may benefit from a research design that matches subjects for age and risk factors to get a more accurate picture in the anger/CHD connection.

*Group Differences Between MAD-AS Subscale Scores*

A multivariate analysis of variance (MANOVA) was conducted on the MAD-AS subscale scores in the cardiac and non-cardiac groups to determine if there were differences. The research hypothesis was that the cardiac group would score higher on the MAD-AS subscales than would the non-cardiac group. This test revealed non-significant results, Wilks Lambda = .968, F (6, 283) = 1.545, $p > .05$. Again, because age effects in anger have been reported (Spielberger, 1999), a multivariate analysis of covariance (MANCOVA) was conducted using age as a covariate. This test was significant, Wilks Lambda = .943, F (6,282) = 2.839, $p < .05$. Significant subscales that were observed were: F2 Verbal Expression F = 3.942; $p < .05$; F3 Behavioral Dyscontrol

$F = 6.021$, $p < .05$; and F4 Physical Aggression $F = 10.188$, $p < .05$. Thus, it would appear that men who have difficulty with more explosive forms of anger (e.g. "I insult people when I am angry", "I throw things when I am angry", "I lose control when I am angry", "When provoked, I hit people") have more incidence of CHD when age is controlled for. These findings are consistent with the findings of the Onset Study (Mittleman, et al., 1995) and the Sheep Study (Moller, Hallqvist, Diderichsen, Theorell, Reuterwall, & Ahlbom, 1999) because intense anger episodes were found to be capable of triggering a cardiac event.

*Treatment Implications*

Mahan (2001) described his rationale for developing the MAD-AS as "the need to create a relatively brief, clinically sensitive, screening instrument for use by cognitive-behavioral therapists interested in the treatment of anger not only for assessing anger but also for measuring treatment outcome." This is an important contribution to the field because anger difficulty is implicated in every major facet of one's life. The potential utilization of this measure to track therapeutic progress would be similar to the use of the Beck inventories because of the brevity and strong psychometric properties of the MAD-AS. Risk assessment and treatment planning could also be accomplished utilizing the MAD-As, given the descriptive data provided in the subscales.

It is quite plausible that the medical community may benefit from utilization of the MAD-AS. Given that the American Heart Association added psychosocial evaluation and intervention into its Core Components of Cardiac Rehabilitation/Secondary

Prevention program (AHA, 2000), it appears logical that evaluation of one's level of anger is as important a measure as evaluation of one's level of stress. Once screening identifies an individual found to have difficulties with anger, tailored interventions could be implemented. The more complete and well-rounded the rehabilitation program, the better likelihood of successful recovery and improved health for countless Americans.

The initiation of screenings and interventions for problematic anger after CHD has occurred is by all accounts a reactive approach, whereas more insurance providers are attempting to become more pro-active in health matters because this is a more fiscally sound approach. Promotion of wellness, of prevention, and of screening that prevents, detects or slows the progression of disease has grown recently (Scala-Foley, Caruso, Archer, & Reinhard, 2004). Accordingly, a new initiative for Medicare in 2005 is a one-time physical wellness exam (as cited in Scala-Foley, Caruso, Archer, & Reinhard, 2004), which will include, among other things, early detection of CHD. This benefit reflects the shift from disease treatment to disease prevention with the recognition of biopsychosocial factors involved in disease processes. It is plausible that those found to be at high risk for CHD through wellness exams could undergo further screening; this could include the MAD-AS in attempts to reduce modifiable risk factors.

*Implications for Future Research*

There are several areas that would benefit from further investigation. First and foremost is the disparity in the experience and expression of anger between the older age

group and the younger participants. A better understanding of how individuals experience and cope with anger as they age would establish a clearer picture of the relationship of anger to CHD and other health concerns. The sample of cardiac patients had a poorer health status than the non-cardiac sample; however, it is unclear exactly what was responsible for this disparity. A study that matches subjects for traditional risk factors would lend a better understanding of potential anger dimensions that accompany the CHD process. This may provide a valuable screening framework for a potential triage of cardiac patients to target at-risk individuals more successfully.

Women were purposely excluded from this study to keep the highest degree of integrity of the anger construct among men entering the age at which CHD is most prevalent. The exclusion was necessitated by observed gender differences in anger experience and expression. For instance, Linden, Hogan, Rutledge, Chawala, Lenz, and Leung (2003) found that women used a wider range of coping strategies than men, especially anger diffusion strategies and social support seeking. This suggests that anger may play a different role in how women perceive, express, and are generally affected by anger. If women indeed seek social support more than men, does this equate to anger reduction or to reduced physiological impact of anger on the body? If so, is this a health-protective factor? Research that examines these gender differences in anger would contribute greatly to the field. Furthermore, a recent study (Eaker, Sullivan, Kelly-Hayes, D'Agostino, & Benjamin, 2004) found that after having controlled for traditional risk factors of CHD, neither the psychosocial variables of trait anger, TABP, nor hostility were related to CHD in women; however, trait anger was found to relate to total

mortality in men. Future research that addresses these issues would be extremely beneficial to understanding and improving women's health.

*Limitations of the Study*

The major limitation of this study is its use of self-report measures. Inherent in self-report measures is the possibility that not all participants were fully accurate in their responses. The use of self-report instruments may be confounded by social desirability effects; this was found by Martin (2002) in a study that looked at self-report scores versus spouse's scores or significant others' scores. This study concluded that the self-reporters were significantly more apt to respond in a manner that was more socially desirable even when the individual would not necessarily act that way in a particular situation. Responses can also be affected by low self-awareness of one's own cognitive, behavioral, and affective responses to anger and hostility (Kneip, Delamater, Ismond, Milford, Salvia, & Schwartz, 1993) because of biases.

Along with self-report limitation is the fact that although a person may be predisposed to react in a certain manner, that does not necessarily mean that he or she will always react that way in all situations. One low in trait anger may react to a stressful situation inconsistently. It also does not mean that individuals experience the same number of stressful situations; this creates a difficulty in predicting the real effect on CHD However, given even the limitations of studying anger and hostility and their

relationships to CHD through self-report measures, it is clear that there is a growing body of evidence that implicates anger in some aspect of CHD.

*Summary*

In summary, this study found psychometric support that the MAD-AS is a structurally durable and stable measurement of the cognitive, affective, and behavioral components of anger. The fact that this is the fourth study to come to the same conclusion bodes well for the instrument. This is a significant contribution to the field because this measurement less lengthy than other instruments and therefore estimated to be less costly. In our present managed-care environment, a time and cost conscious screening measure that is also psychometrically sound would prove quite useful.

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Appendix A

Dear Participant:

We are doing a study on the relationship between feelings and medical problems. If you are a male between the ages of 35 and 63 and you have no other major medical illnesses other than a diagnosis of a heart ailment (such as cancer or organ failure) and can read and speak English, you may be eligible to be in the study. Your decision to be in this study is completely voluntary. You may decide not to participate or discontinue participation at any time. In no way will your health care be affected whether or not you choose to be in the study. All information will be kept strictly confidential. You will not be asked to provide your name on any material, therefore, no one will be able to identify you. Your physician and health care workers will not have access to this information.

If you choose to be part of this study, you will be given a packet and asked to fill out three questionnaires that take about 20 minutes of your time. The first questionnaire asks about your age, sex, marital status, lifestyle, and health history. The other two questionnaires ask questions about your feelings. If your packet contains two sets of questionnaires, you are requested to fill out one packet now, and the one marked "RETEST" **ONE WEEK** later and place it in the postage paid envelope provided and mail it back to the researcher. There will be a number on each form that is used to match forms should pages get separated and to match mailed in retest packets.

The questionnaires ask about your thoughts, feelings, and behaviors. It is possible that you may learn something about yourself of which you did not know before. In the unlikely event that you become uncomfortable or upset with your answers to any of these questions, please contact First Hospital Wyoming Valley at 1(800) 624-9902 if you are in Luzerne county or Scranton Counseling Center at 1(570) 348-6100 if you are in Lackawanna county for a mental health referral. You may even choose to contact the principal investigator, Robert A. DiTomasso, Ph.D., A.B.P.P. at (215) 871-6511.

If you would like a summary of the results of this study, you may contact the co-investigator, Kimberly D'Andrea, M.S. via e-mail at KimberlyDa.studpob.Stud.

Thank you very much for your participation in this investigation.

Kimberly S. D'Andrea, M.S.
Philadelphia College of Osteopathic Medicine
Department of Psychology
4190 City Avenue
Philadelphia, PA 19131

Robert A. DiTomasso, Ph.D., A.B.P.P.
Professor and Vice Chair, Department of Psychology
Philadelphia College of Osteopathic Medicine
4190 City Avenue
Philadelphia, Pa 19131

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Philadelphia, PA 19131

Robert A. DiTomasso, Ph.D., A.B.P.P.
Professor and Vice Chair, Department of Psychology
Philadelphia College of Osteopathic Medicine
4190 City Avenue
Philadelphia, Pa 19131

Appendix A

# DEMOGRAPHIC SHEET

## PLEASE COMPLETE EACH QUESTION BY FILLING OUT OR CHECKING THE APPROPRIATE ANSWER

1) **Age:** _____
2) **Sex:** Male_____ Female_____
3) **Marital Status:** Single_____ Married_____ Separated_____
Divorced_____ Widowed_____ Co-Habitating_____

**Cardiac History:**

4) Have you ever been diagnosed with a cardiac illness? Yes_____ No_____
5) Do you have a heart condition that you had since birth? Yes_____ No_____
6) If you are currently taking medication for a cardiac diagnosis, check the ones you take:
_____calcium channel blockers ( e.g.: Procardia/Nifedipine, Norvasc/Amlodipine, Calan/Verapimil)
_____ACE inhibitors (e.g. Capoten/Captopril, Vasotec/Enalapril, Prinivil/Lisinopril, Altace/Ramipril)
_____beta-blockers (e.g. Metaprolol/Lopressor/Toprol, Attenolol/Tenormin, Propranolol/Inderal)
_____diuretics (water pill)
_____aspirin

**General Medical History:**

7) Do you have diabetes? Yes_____ No_____
8) Do you have high blood pressure? Yes_____ No_____
9) If yes, are you taking medication to lower it? (e.g. Cozaar/Losartan, Diovan/Valsartan, Avapro/Irbpsartan, Atacand/Candesartan) Yes_____ No_____
10) Do you have high cholesterol? Yes_____ No_____
11) If yes, what is your total cholesterol level? Less than 200_____ 200 – 239_____ Above 240 _____
12) Do you smoke? Yes_____ No_____
13) If yes, how much? Less than 1 pack per day_____ More than 1 pack a day_____
14) Do you exercise? Yes_____ No_____
15) If yes, is it vigorous or moderate?
_____Vigorous (activity causing hard breathing and sweating for at least 20 minutes on 3 or more of 7 days)
_____Moderate (activities such as walking or bicycling lasting at least 30 minutes on 5 or more of 7 days)
16) Are you overweight? Yes _____ No_____
17) If yes, how much? 10–20lbs_____ 20–30lbs_____ Over 30 lbs_____
18) Do you have a family history of heart disease? Yes_____ No_____
19) Are you currently receiving psychological counseling? Yes_____ No_____
20) Are you currently taking antidepressant medication? (e.g.: Prozac, Zoloft, Paxil, Wellbutrin) Yes_____ No_____
21) Are you currently taking antianxiety medication? (e.g.: Xanax, Valium, Ativan) Yes_____ No_____
22) Do you have any major illnesses? (cancer, organ failure) Yes_____ No_____
23) Have you been diagnosed as having TMJ or teeth grinding? Yes_____ No_____

Appendix A

## DEMOGRAPHIC SHEET

## LEASE COMPLETE EACH QUESTION BY FILLING OUT OR CHECKING THE APPROPRIATE ANSWER

1) **Age:** _____
2) **Sex:** Male _____ Female_____
3) **Marital Status:** Single_____ Married_____ Separated_____
Divorced_____ Widowed_____ Co-Habitating_____

**General Medical History:**

4) Have you ever been diagnosed with a cardiac illness? Yes_____ No_____
5) Do you have diabetes? Yes_____ No_____
6) Do you have high blood pressure? Yes_____ No_____
7) If yes, are you taking medication to lower it? (e.g.: Cozaar/Losartan, Diovan/Valsartan, Avapro/Irbpsartan, Atacand/Candesartan) Yes_____ No_____
8) Do you have high cholesterol? Yes_____ No_____
9) If yes, what is your total cholesterol level? Less than 200_____200 – 239_____
240 or above_____
10) Do you smoke? Yes_____ No_____
11) If yes, how much? Less than 1 pack per day_____More than 1 pack a day_____
12) Do you exercise? Yes_____ No_____
13) If yes, is it vigorous or moderate?
_____Vigorous (activity causing hard breathing and sweating for at least 20 minutes on 3 or more of 7 days)
_____Moderate (activities such as walking or bicycling lasting at least 30 minutes on 5 or more of 7 days)
14) Are you overweight? Yes _____ No_____
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18) Are you currently taking antianxiety medication? (e.g.: Xanax, Valium, Ativan) Yes_____ No_____
19) Do you have any other major illnesses? (e.g. cancer, organ failure) Yes_____ No_____
20) Have you been diagnosed as having TMJ or teeth grinding? Yes_____ No_____

Appendix A

# MAD-AS

**Marital Status:** _____ **Age:**_____ **Sex:** _____

This questionnaire consists of 43 statements or quartets. After reading each group of statements carefully circle the number (0, 1, 2 or 3) next to the **one** statement in each group which best describes the way you have been feeling the past week including today. There are no right or wrong answers. Carefully read each question before answering.

---

1. 0 I **never** feel a need to get even with those who anger me.
   1 I **sometimes** feel a need to get even with those who anger me.
   2 I **often** feel a need to get even with those who anger me.
   3 I **always** feel a need to get even with those who anger me.

2. 0 My anger **never** keeps me up at night.
   1 My anger **sometimes** keeps me up at night.
   2 My anger **often** keeps me up at night.
   3 My anger **always** keeps me at night.

3. 0 I **never** have trouble letting go of my anger.
   1 I **sometimes** have trouble letting go of my anger.
   2 I **often** have trouble letting go of my anger.
   3 I **always** have trouble letting go of my anger.

4. 0 I **never** anger more frequently than most people.
   1 I **sometimes** anger more frequently than most people.
   2 I **often** anger more frequently than most people.
   3 I **always** anger more frequently than most people.

5. 0 I **never** get angry without reason.
   1 I **sometimes** get angry without reason.
   2 I **often** get angry without reason.
   3 I **always** get angry without reason.

6. 0 I am **never** quick to anger.
   1 I am **sometimes** quick to anger.
   2 I am **often** quick to anger.
   3 I am **always** quick to anger.

**Continued on next page ⇒**

7. 0 I **never** have trouble letting go of things that have angered me in the past.
   1 I **sometimes** have trouble letting go of things that have angered me in the past.
   2 I **often** have trouble letting go of things that have angered me in the past.
   3 I **always** have trouble letting go of things that have angered me in the past.

8. 0 I **never** hold grudges against those who have angered me.
   1 I **sometimes** hold grudges against those who have angered me.
   2 I **often** hold grudges against those who have angered me.
   3 I **always** hold grudges against those who have angered me.

9. 0 I **never** lose control when angry.
   1 I **sometimes** lose control when angry.
   2 I **often** lose control when angry.
   3 I **always** lose control when angry.

10. 0 I **never** throw things when I am angry.
    1 I **sometimes** throw things when I am angry.
    2 I **often** throw things when I am angry.
    3 I **always** throw things when I am angry.

11. 0 I can **never** control my temper.
    1 I can **sometimes** control my temper.
    2 I can **often** control my temper.
    3 I can **always** control my temper.

12. 0 I **never** hit those who anger me.
    1 I **sometimes** hit those who anger me.
    2 I **often** hit those who anger me.
    3 I **always** hit those who anger me.

13. 0 I am **never** a hot head.
    1 I am **sometimes** a hot head.
    2 I am **often** a hot head.
    3 I am **always** a hot head.

14. 0 I am **never** critical of others when angry.
    1 I am **sometimes** critical of others when angry.
    2 I am **often** critical of others when angry.
    3 I am **always** critical of others when angry.

**Continued on next page ⇒**

15. 0 I **never** argue with people without reason.
    1 I **sometimes** argue with people without reason.
    2 I **often** argue with people without reason.
    3 I **always** argue with people without reason.

16. 0 I **never** blame others for my anger.
    1 I **sometimes** blame others for my anger.
    2 I **often** blame others for my anger.
    3 I **always** blame others for my anger.

17. 0 I **never** think about things that anger me.
    1 I **sometimes** think about things that anger me.
    2 I **often** think about things that anger me.
    3 I **always** think about things that anger me.

18. 0 When I am angry people **never** fear me.
    1 When I am angry people **sometimes** fear me.
    2 When I am angry people **often** fear me.
    3 When I am angry people **always** fear me.

19. 0 When I am angry I **never** have thoughts of hurting others.
    1 When I am angry I **sometimes** have thoughts of hurting others.
    2 When I am angry I **often** have thoughts of hurting others.
    3 When I am angry I **always** have thoughts of hurting others.

20. 0 People **never** intend to anger me.
    1 People **sometimes** intend to anger me.
    2 People **often** intend to anger me.
    3 People **always** intend to anger me.

21. 0 My anger **never** caused me problems in my relationships.
    1 My anger **sometimes** caused me problems in my relationships.
    2 My anger **often** caused me problems in my relationships.
    3 My anger **always** caused me problems in my relationships.

22. 0 My anger has **never** caused me problems on the job.
    1 My anger has **sometimes** caused me problems on the job.
    2 My anger has **often** caused me problems on the job.
    3 My anger has **always** caused me problems on the job.

**Continued on next page ⇒**

23. 0 The behavior of others **never** causes me to get angry.
    1 The behavior of others **sometimes** causes me to get angry.
    2 The behavior of others **often** causes me to get angry.
    3 The behavior of others **always** causes me to get angry.

24. 0 After expressing my anger I **never** feel guilty.
    1 After expressing my anger I **sometimes** feel guilty.
    2 After expressing my anger I **often** feel guilty.
    3 After expressing my anger I **always** feel guilty.

25. 0 I **never** tolerate others mistakes.
    1 I **sometimes** tolerate others mistakes.
    2 I **often** tolerate others mistakes.
    3 I **always** tolerate others mistakes.

26. 0 I **never** insult people when I am angry.
    1 I **sometimes** insult people when I am angry.
    2 I **often** insult people when I am angry.
    3 I **always** insult people when I am angry.

27. 0 When angry, I **never** let it show.
    1 When angry, I **sometimes** let it show.
    2 When angry, I **often** let it show.
    3 When angry, I **always** let it show.

28. 0 I **never** lose control when angry.
    1 I **sometimes** lose control when angry.
    2 I **often** lose control when angry.
    3 I **always** lose control when angry.

29. 0 I **never** threaten people when angry.
    1 I **sometimes** threaten people when angry.
    2 I **often** threaten people when angry.
    3 I **always** threaten people when angry.

30. 0 I am **never** argumentative.
    1 I am **sometimes** argumentative.
    2 I am **often** argumentative.
    3 I am **always** argumentative.

**Continued on next page ⇒**

31. 0 I **never** tell people when they annoy me.
    1 I **sometimes** tell people when they annoy me.
    2 I **often** tell people when they annoy me.
    3 I **always** tell people when they annoy me.

32. 0 When people disagree with me, I **never** argue.
    1 When people disagree with me, I **sometimes** argue.
    2 When people disagree with me, I **often** argue.
    3 When people disagree with me, I **always** argue.

33. 0 I **never** feel bitter about things.
    1 I **sometimes** feel bitter about things.
    2 I **often** feel bitter about things.
    3 I **always** feel bitter about things.

34. 0 When provoked, I **never** hit people.
    1 When provoked, I **sometimes** hit people.
    2 When provoked, I **often** hit people.
    3 When provoked, I **always** hit people.

35. 0 When under stress, I **never** get angry.
    1 When under stress, I **sometimes** get angry.
    2 When under stress, I **often** get angry.
    3 When under stress, I **always** get angry.

36. 0 Once angered, I **never** get over it quickly.
    1 Once angered, I **sometimes** get over it quickly.
    2 Once angered, I **often** get over it quickly.
    3 Once angered, I **always** get over it quickly.

37. 0 I **never** feel a sense of relief after an angry outburst.
    1 I **sometimes** feel a sense of relief after an angry outburst.
    2 I **often** feel a sense of relief after an angry outburst.
    3 I **always** feel a sense of relief after an angry outburst.

38. 0 When angry, I **never** feel my heart beating faster.
    1 When angry, I **sometimes** feel my heart beating faster.
    2 When angry, I **often** feel my heart beating faster.
    3 When angry, I **always** feel my heart beating faster.

**Continued on next page ⇒**

39. 0 When angry, my muscles **never** feel tense.
    1 When angry, my muscles **sometimes** feel tense.
    2 When angry, my muscles **often** feel tense.
    3 When angry, my muscles **always** feel tense.

40. 0 When angry, my breathing is **never** rapid.
    1 When angry, my breathing is **sometimes** rapid.
    2 When angry, my breathing is **often** rapid.
    3 When angry, my breathing is **always** rapid.

41. 0 When angry, I **never** feel restless or agitated.
    1 When angry, I **sometimes** feel restless or agitated.
    2 When angry, I **often** feel restless or agitated.
    3 When angry, I **always** feel restless or agitated.

42. 0 When someone offends me I **never** retaliate.
    1 When someone offends me I **sometimes** retaliate.
    2 When someone offends me I **often** retaliate.
    3 When someone offends me I **always** retaliate.

43. 0 In difficult situations, I **never** get angry.
    1 In difficult situations, I **sometimes** get angry.
    2 In difficult situations, I **often** get angry.
    3 In difficult situations, I **always** get angry.

---

# Appendix A

# STAXI-2™

## Item Booklet (Form HS)

### Instructions

In addition to this Item Booklet you should have a STAXI-2 Rating Sheet. Before beginning, enter your name, gender, and age; today's date; years of education completed, your marital status, and your occupation in the spaces provided at the top of the STAXI-2 Rating Sheet.

This booklet is divided into three Parts. Each Part contains a number of statements that people use to describe their feelings and behavior. Please note that each Part has *different* directions. Carefully read the directions for each Part before recording your responses on the Rating Sheet.

There are no right or wrong answers. In responding to each statement, give the answer that describes you best. DO NOT ERASE! If you need to change your answer, mark an "X" through the incorrect response and then fill in the correct one.

**Examples**

1. ①  ④

2. ①  ③ ④

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## Part 1 Directions

A number of statements that people use to describe themselves are given below. Read each statement and then blacken the appropriate circle on the Rating Sheet to indicate how you feel *right now*. There are no right or wrong answers. Do not spend too much time on any one statement. Mark the answer that *best* describes your *present feelings*.

| Fill in ① for *Not at all* | Fill in ② for *Somewhat* | Fill in ③ for *Moderately so* | Fill in ④ for *Very much so* |
|---|---|---|---|

### How I Feel Right Now

1. I am furious
2. I feel irritated
3. I feel angry
4. I feel like yelling at somebody
5. I feel like breaking things
6. I am mad
7. I feel like banging on the table
8. I feel like hitting someone
9. I feel like swearing
10. I feel annoyed
11. I feel like kicking somebody
12. I feel like cursing out loud
13. I feel like screaming
14. I feel like pounding somebody
15. I feel like shouting out loud

## Part 2 Directions

Read each of the following statements that people have used to describe themselves, and then blacken the appropriate circle to indicate how you *generally* feel or react. There are no right or wrong answers. Do not spend too much time on any one statement. Mark the answer that *best* describes how you *generally* feel or react.

| Fill in ① for *Almost never* | Fill in ② for *Sometimes* | Fill in ③ for *Often* | Fill in ④ for *Almost always* |
|---|---|---|---|

### How I Generally Feel

16. I am quick tempered
17. I have a fiery temper
18. I am a hotheaded person
19. I get angry when I'm slowed down by others' mistakes
20. I feel annoyed when I am not given recognition for doing good work
21. I fly off the handle
22. When I get mad, I say nasty things
23. It makes me furious when I am criticized in front of others
24. When I get frustrated, I feel like hitting someone
25. I feel infuriated when I do a good job and get a poor evaluation

## Part 3 Directions

Everyone feels angry or furious from time to time, but people differ in the ways that they react when they are angry. A number of statements are listed below which people use to describe their reactions when they feel *angry* or *furious*. Read each statement and then blacken the appropriate circle to indicate how *often* you *generally* react or behave in the manner described when you are feeling angry or furious. There are no right or wrong answers. Do not spend too much time on any one statement.

| Fill in ① for *Almost never* | Fill in ② for *Sometimes* | Fill in ③ for *Often* | Fill in ④ for *Almost always* |
|---|---|---|---|

### How I Generally React or Behave When Angry or Furious...

26. I control my temper
27. I express my anger
28. I take a deep breath and relax
29. I keep things in
30. I am patient with others
31. If someone annoys me, I'm apt to tell him or her how I feel
32. I try to calm myself as soon as possible
33. I pout or sulk
34. I control my urge to express my angry feelings
35. I lose my temper
36. I try to simmer down
37. I withdraw from people
38. I keep my cool
39. I make sarcastic remarks to others
40. I try to soothe my angry feelings
41. I boil inside, but I don't show it
42. I control my behavior
43. I do things like slam doors
44. I endeavor to become calm again
45. I tend to harbor grudges that I don't tell anyone about
46. I can stop myself from losing my temper
47. I argue with others
48. I reduce my anger as soon as possible
49. I am secretly quite critical of others
50. I try to be tolerant and understanding
51. I strike out at whatever infuriates me
52. I do something relaxing to calm down
53. I am angrier than I am willing to admit
54. I control my angry feelings
55. I say nasty things
56. I try to relax
57. I'm irritated a great deal more than people are aware of

Appendix A

# NOTICE OF RESEARCH PARTICIPATION

The Wyoming Valley Heart Group is currently participating in a research study conducted by Philadelphia College of Osteopathic Medicine. The study is investigating the relationship between feelings and medical problems. If you are a male between the ages of 35 and 63 and have no other major medical conditions (such as cancer or organ failure) you may be able to participate. The choice to be in this study is completely voluntary and all information is anonymous. You will not be identified in any way, your health care providers will not have access to the information. In no way will your health care be affected whether or not you choose to be in the study. You could discontinue your participation at any time without consequences. If you decide to participate you will be asked to fill out three (3) questionnaires that will take about 20 minutes of your time. You may be asked to complete the same questionnaires one week later. Please notify the office staff if you are interested in participating.

Appendix A

# NOTICE OF RESEARCH PARTICIPATION

Dr. Frank Cianci is currently participating in a research study conducted by researchers at Philadelphia College of Osteopathic Medicine. The study is investigating the relationship between feelings and medical problems. If you are a male between the ages of 35 and 63 and have no major medical conditions (such as cancer, heart disease, or organ failure) you may be able to participate. The choice to be in this study is completely voluntary and all information is anonymous. You will not be identified in any way, your health care providers will not have access to the information. In no way will your health care be affected whether or not you choose to be in the study. You could discontinue your participation at any time without consequences. If you decide to participate you will be asked to fill out three (3) questionnaires that will take about 20 minutes of your time. You may be asked to complete the same questionnaires one week later. Please notify the office staff if you are interested in participating.

Appendix A

# SCRIPT FOR PARTICIPATION SOLICITATION

Thank you for your interest in participating in this research study on the relationship between feelings and medical problems conducted by researchers at Philadelphia College of Osteopathic Medicine. You may be included in the study if you are a male between the ages of 35 and 63 and you have no other major medical illnesses other than a diagnosis of a heart ailment and have no psychological problems for which you are currently receiving treatment. Your decision to be in this study is completely voluntary. You may decide not to participate or discontinue participation at any time. In no way will your health care be affected whether or not you choose to be in the study. All information will be kept strictly confidential. You will not be asked to provide your name on any material, therefore, no one will be able to identify you. Your physician and health care workers will not have access to this information.

If you choose to be part of this study, you will be given a packet and asked to fill out three questionnaires that take about 20 minutes of your time. The first questionnaire asks about your age, sex, marital status, lifestyle, and health history. The other two questionnaires ask questions about your feelings. If your packet contains two sets of questionnaires, you are requested to fill out one packet now, and the one marked "RETEST" **ONE WEEK** later and place it in the postage paid envelope provided and mail it back to the researcher. There will be a number on each form that is used to match forms should pages get separated and to match mailed in re-test packets.

The questionnaires ask about your thoughts, feelings, and behaviors. It is possible that you may learn something about yourself of which you did not know before. In the unlikely event that you become uncomfortable or upset with your answers to any of these questions, please refer to the Introduction letter that contains two local mental health referral telephone numbers. You may even choose to contact the principal investigator at the number listed on the Introduction Letter or receive a summary of the results of the study at the E-mail address listed on the Introduction Letter.

Thank you very much for you participation in this study.

Appendix A

# SCRIPT FOR PARTICIPATION SOLICITATION

Thank you for your interest in participating in this research study on the relationship between feelings and medical problems conducted by researchers at Philadelphia College of Osteopathic Medicine. You may be included in the study if you are a male between the ages of 35 and 63 and you have no major medical illnesses (such as cancer, heart disease, organ failure) and have no psychological problems for which you are currently receiving treatment. Your decision to be in this study is completely voluntary. You may decide not to participate or discontinue participation at any time. In no way will your health care be affected whether or not you choose to be in the study. All information will be kept strictly confidential. You will not be asked to provide your name on any material, therefore, no one will be able to identify you. Your dentist and health care workers will not have access to this information.

If you choose to be part of this study, you will be given a packet and asked to fill out three questionnaires that take about 20 minutes of your time. The first questionnaire asks about your age, sex, marital status, lifestyle, and health history. The other two questionnaires ask questions about your feelings. If your packet contains two sets of questionnaires, you are requested to fill out one packet now, and the one marked "RETEST" **ONE WEEK** later and place it in the postage paid envelope provided and mail it back to the researcher. There will be a number on each form that is used to match forms should pages get separated and to match mailed in retest packets.

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Appendix B

**Commitment That Trained Office Staff Members Who Collect Data Will Abide By All Rules of Confidentiality**

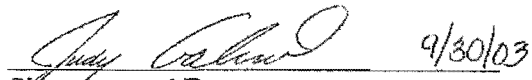
I understand that no participant in this research study will be identified. I commit to abiding by all rules of confidentiality in ensuring that all participants will remain anonymous and all information will remain confidential.

Kiren Lynch 10/3/03
Signature and Date

Appendix B

**Commitment That All Who Collect Data Will Abide By All Rules of Confidentiality**

I understand that no participant in this research study will be identified. I commit to abiding by all rules of confidentiality in ensuring that all participants will remain anonymous and all information will remain confidential.



Signature and Date

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Donna Topp RN 9/30/03

Signature and Date

Appendix B

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Michele Abramski RN 9-30-03
Signature and Date

# Appendix B

KIMBERLY S. D'ANDREA, M.S.
5 ALCOTT CLOSE
MOOSIC, PA 18507
PHONE: 570.344.5599 FAX: 570.344.6699 E-MAIL: KimJeep88@[illegible]

September 16, 2003

Robert A. DiTomasso, Ph.D., A.B.P.P.
Professor and Vice Chair, Department of Psychology
Philadelphia College of Osteopathic Medicine
4190 City Avenue
Philadelphia, Pa 19131

James P. Mahan, Psy.D.
6 Barley Court
Marlton, NJ 08053

Dear Drs. DiTomasso and Mahan,

I am currently a Psy.D. Candidate at Philadelphia College of Osteopathic Medicine. I am writing to you today to respectfully request permission to utilize the Mahan and DiTomasso Anger Scale (MAD-AS) as one of the instruments in my doctoral dissertation. My study is titled "A normative study of the Mahan and DiTomasso Anger Scale (MAD-AS) in an outpatient cardiac population" and it seeks to investigate the psychometric properties of the MAD-AS in an outpatient cardiac population. I also hope to add to the growing support for the correlation between trait anger and coronary heart disease. I would be happy to share with you the results of my investigation. Please indicate if you grant me permission to use the MAD-AS by signing below and placing this letter in the enclosed addressed and stamped envelope. I look forward to your reply. Thank you very much.

Sincerely,

Kimberly S. D'Andrea, M.S.

Robert A. DiTomasso, Ph.D., A.B.P.P.

# Appendix B

KIMBERLY S. D'ANDREA, M.S.
5 ALCOTT CLOSE
MOOSIC, PA 18507
PHONE: 570.344.3599 FAX: 570.344.6699 E-MAIL: KIMJDA88@WILKES.EDU

September 16, 2003

Robert A. DiTomasso, Ph.D., A.B.P.P.
Professor and Vice Chair, Department of Psychology
Philadelphia College of Osteopathic Medicine
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Philadelphia, Pa 19131

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